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THE POTENTIALITIES OF PREVENTIVE GERIATRICS*

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GERONTOLOGY is the science of aging. Geriatrics may be defined as that special field of medical practice dealing with disease in aged persons. The two must not be confused; the aged are the consequences of aging. The aged are people; aging is a process. Aging begins with the conception of a new individual and continues throughout life. Aging is a part of living, and thus involves both evolutional and involutional phenomena. Senescence may be defined as that part of the aging process which occurs after the peak of development. Although the changes of senescence are largely involutional, it must not be assumed that all the consequences represent decline. There are important compensatory increments in certain functional capacities. Though long neglected, the unutilized potentialities of the aging and aged are worthy of serious consideration.

Just as geriatrics is concerned with the treatment of disease, preventive geriatrics is interested in the prevention of disease in later life. Probably the most significant period from the viewpoint of prophylactic geriatrics is the two decades from forty to sixty. It is in this period, or even before, that the involutional processes start, later to reveal themselves in the more obvious evidences of aging. Preventive geriatrics does not set as its objective the prevention of aging. To do that would be to arrest life. We cannot stop growing older. But we can hope to modify the consequences of aging and retard the progression of certain factors that make for premature senescence. Although aging is inevitable, the character of growing older is perhaps amenable to modification.

Why is the study of aging so young a science? Surely aging is no new phenomenon. It has continued since the beginning of Time. The age of the earth has long interested geologists, the age of the universe has puzzled astronomers, and the age of man as a species has been the concern of anthropologists and archaeologists for many years. But the aging of *man as an individual* has received scant attention, and very little, indeed, is known anent the basic mechanisms of aging as a biologic process. A young world is interested in youth. Mankind is slowly progressing toward maturation. As culture advances, we are commencing to realize the importance of the elderly.

Although the ancient Greeks delighted in the philosophic discussions of their aged seers, in those days the old were objects of curiosity because of their rarity. In Rome, Cicero wrote his famed *De Senectute* several years before his death at sixty-three. He considered himself an old man in the fifties, and justly so, for few of his contemporaries survived to such ripeness. The average life expectancy at birth for the Roman citizen was about twenty-three years, according to cautious estimates. During the next nineteen centuries, life expectancy increased very slowly. Data from New England in 1850 indicate that life expectancy at birth was but forty years. By 1900, this had risen to forty-eight years for the United States as a whole, and since then the rise has been dramatic. In 1930, life expectancy at birth had increased to about sixty years of age, and it is now over sixty-three for white members of the population.

Looking at the changing picture of humanity from another angle, one sees that in 1900 only 17 per cent of the total population of the United States were forty-five years or older. In 1940, 26.5 per cent were over forty-five, and conservative projection results in the estimate that in 1980 — only another forty years hence — more than 40 per cent of the population will be over forty-five years old. Recently released figures from the 1940 census reveal that the median age of the population of this country increased from slightly more than twenty-six years in 1930 to just under twenty-nine years in 1940. This is an increase

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of two and a half years of median age within a decade. At that rate, the median age of the population will be forty-four years in another half century. Furthermore, the number of persons aged sixty-five or more has increased from 6,633,805 to 8,956,206. This is a rise of 36.5 per cent among the elderly, as contrasted with a 7.2 per cent increase in the total population in the last decade.

Such figures speak for themselves. Gerontology is no longer merely academically interesting, but has become an urgent matter in the minds of those sufficiently farsighted to see the handwriting on the wall. The nation is aging rapidly. The virile, violent but short-lived days of physical pioneering are largely past. The future holds promise of profound change. A period of intellectual conquest may be dawning. Man at last lives long enough to have time to think.

The situation is wholly without precedent. Never before in the history of mankind has a like problem presented itself; there are no marked trails to follow. We are almost as ignorant of the potentialities of a maturer mankind as Columbus was of the vast future of this continent. As explorers, we can see but the fringe of a new era, a period fraught with undefined menaces, but likewise presenting vistas of delightful aspect. The children of today will be the elderly of tomorrow. It is our responsibility to explore so far as we are able, to light their way and warn them of dangers, so that this apparent boon of increased longevity does not become a curse. For great longevity without health is not only an individual tragedy, but may develop into a social evil viciously destructive to national economy. Contrariwise, increased longevity of the population can become an incalculably valuable asset to any commonwealth if the potentialities of the elderly are wisely guided.

Urgent and unanswered problems arise in economic, sociologic, political and psychiatric fields as well as in medicine. The direction of some of these problems is already indicated. The clamor of the aged for economic security is heard throughout the breadth of the land. The clamor will become louder. Fantastic schemes are advocated with fanatic vigor and have already taken on political flavor. The problem of employment of older men demands serious and immediate attention, for logic inexorably leads to the conclusion that either the increasing millions of elderly people must have opportunity to work and support themselves or the proportionately dwindling group of younger persons will have to support them in one way or another. One answer implies productivity suited to capacity, the other destructive costs on what may ultimately become a minority.

Education, which is preparation, has not kept pace with these changes in the social order. Educational curriculums are still geared to the day when life expectancy was fifteen or twenty years less than it is today, when it sufficed that education prepare the boy or girl for the competition of adult life. Neither parents nor teachers have taken cognizance of the necessity for preparation for old age. It has been assumed with complacent smugness that the adult would learn how to grow old — gracefully, happily and usefully — without training or aid. Unfortunately, very few learn this spontaneously. The time has come when educators must revise their objectives. The accomplishments of such men as Oliver Wendell Holmes, William H. Welch, Goethe, Edison, Titian and many others in the evening of their lives are mere indicators of the vast storehouse of latent treasure in those so often sneeringly dubbed "old men." Such careers are rarely fortuitous. Continued development is greatly enhanced by cultivation.

The remarkable increase in life expectancy is attributable largely to control of infectious diseases in childhood and early adult life. This has been accomplished chiefly by the public-health officer and the pediatrician. Through the institution and maintenance of modern sanitation, infant mortality has been dramatically reduced. So-called "summer complaint" was a dreaded disorder of infants that only the older ones among us remember. It passed with the disappearance of contaminated milk and water. Typhoid fever not so many years ago filled the wards of the hospitals in our major cities. Today, it is difficult to find sufficient cases for the teaching of medical students. Cholera, plague, smallpox, diphtheria and scarlet fever are under control through quarantine and immunization. Today, an epidemic of any of these diseases would bring about an immediate condemnation of the health department of the afflicted community. The maintenance of such safety is dependent on unrelaxing vigilance and constant educational activity. The daily hygiene and the nutrition of the people likewise improve under the guidance of continuous dissemination of the knowledge gained by research.

Prophylactic pediatrics has contributed greatly to increasing life expectancy. Not only has there been a vast improvement in the curative therapy of pediatric disease, but the pediatricians have done more than any other group of physicians to advocate and apply individual preventive medicine. By better feeding of presumably healthy ba-

bies and by prophylactic immunization, they have made healthy children healthier. The pediatrician has recognized that health is more than the mere absence of disease, that there are degrees of health. Such prophylactic practices have not only reduced infant and juvenile mortality, but have also unquestionably prevented many chronic sequelae from childhood ills. It is worthy of emphasis to note that marked advances in pediatric knowledge followed recognition of the fundamental concept that the child is not merely "the little man," but presents nutritional, immunologic, functional and structural problems and characteristics peculiar to infancy or childhood or both. A similar focus of attention in connection with senescence presents quite different problems because of specific, though ill understood and less appreciated, structural and functional differences. Thus geriatrics, or the care of the aged, becomes dependent on gerontology, the science of aging.

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Before discussing the application and potentialities of preventive geriatrics, it might be well to orient the problems of gerontology, which are logically divisible into three major categories.

The first is the biology of senescence as a process. Aging is a part of living. The basic biologic processes involve all different forms of life, and should therefore be amenable to study by many disciplines. It must be admitted that ignorance of this subject is profound. Unanswered as yet are such fundamental questions as just what happens to a cell with aging, why aging occurs, what accelerates or retards it, what mechanisms are involved, and why. The elucidation of these basic questions may solve many riddles, among them the riddle of cancer and perhaps that of arteriosclerosis. Scientifically, the cancer problem is but a subdivision of the larger question of aging.

The second category includes the clinical problems of senescence in man, which are clearly divisible into those relating to normal and to abnormal senescence, the latter conditioned by the disorders associated with advancing years.

Normal aging brings many changes, some obvious, others obscure, but all insidious and inevitably progressive. Structural alterations, psychologic changes and biochemical and physiologic differences arise. Normal is not a fixed point but a series of variables that change with age. Chronologic age, as measured in years and months, is not identical with biologic age. Physiologic age varies with each person. The greater the duration of life, the greater the variation. Furthermore, no one ages uniformly throughout, for different structures and systems age at different rates at various times in the life span. At certain ages, the involution of certain structures apparently becomes accelerated. This is illustrated by the rapid thymic involution in infancy and by the quickened regression of the generative organs at the climacteric. Mental aging likewise reveals much individual variation. Because of the complex relation, no single criterion of physiologic age can ever prove feasible.

There is a common misconception that senescence implies decline alone. This is distinctly an erroneous concept, for there is considerable compensatory enhancement of certain functional capacities. For example, loss of physical strength and speed of reaction are often counterbalanced by increased skill and judgment. It has been said that the older mind does not learn readily. The popular phrase, "You can't teach an old dog new tricks," has done immeasurable harm, for it has become so fixed in the minds of young and old alike that its validity is assumed. As a result, many older people admit defeat before trying, and opportunities for adult education are suppressed. Recent and comprehensive studies reveal that once this adult resistance to learning is overcome, the capacity to learn is but very slightly diminished by aging. There is some slowing of the rate but an increase in thoroughness. It would be better to reiterate "It is never too late to learn."

There are many disorders the frequency of which rises sharply with advancing years. The distinctions between the changes of normal senescence and certain of these so-called "degenerative disorders" are not sharply defined. It is most difficult to distinguish certain phenomena of disease from those attributable to aging. The phenomena of disease are, after all, exaggerations of normal reactions and do not imply new mechanisms.

The most significant of these geriatric disorders are cardiovascular-renal disease, arthritis, diabetes mellitus, gout, cancer and certain syndromes of the climacteric. Of these, the cardiovascular group, including hypertensive arterial disease and arteriosclerosis, is by far the most significant. Arthritis exacts an immense toll of disability, although its mortality is low. A glance at mortality tables reveals that these diseases so frequent in the latter half of life are replacing tuberculosis, accidents, diarrhea and enteritis as the leading causes of death.

Two characteristics common to all these geriatric disorders are of special concern. First, acute infection plays a negligible role in their etiology. Although chronic foci of infection have long been incriminated as contributing

to the causation of arthritis and play a part in the pathogenesis of certain circulatory and renal diseases, none of these disorders can be classified as infectious or communicable. Secondly, all are chronic and progressive, and usually insidious of onset. The progression is often slow, but it is, nevertheless, inevitably persistent. None are self-limited diseases tending toward spontaneous cure and followed by a protracted period of lowered vulnerability. Rather does the progression of these disorders continuously increase vulnerability to exacerbation and accelerated decline. Cure is, at present, largely beyond hope. The best therapeutics cannot cure hypertensive disease, diabetes, gout or chronic arthritis. Control and retardation of progression are, however, feasible. The adequately controlled diabetic patient is vigorous, active, useful and productive despite the fact that he still has the disease. The earlier in the course of these diseases control measures are instituted, the more effective is the therapy. More can be accomplished with the aging than for the aged.

The problems presented to preventive medicine in the later decades of life differ radically from those encountered with younger age groups. These disorders are not amenable to mass prevention as infectious diseases are. They are not contagious. They are progressive. Thus, preventive geriatrics must become individualized, and it must be applied almost continuously. We cannot immunize a person against hypertensive disease and advise that he report again in two years for revaccination. Furthermore, each case of hypertension requires different therapy for control. No two cases are alike. Individualization must be the keystone of the arch of personal preventive medicine. The control of these maladies requires a totally different approach, and sooner or later these problems will absorb a great part of the time and energies of health officer and practitioner.

The third category concerns the socioeconomic problems introduced by increased longevity, greater life expectancy and the rising median age of the population; they are immense and extremely complex. Industry is just awakening to the implications of the fact that the average age of employees is increasing at a surprising rate. This has been occurring despite the fact that until very recently the vast numbers of unemployed permitted and encouraged the selection of younger workers. In the present emergency, the shortage of trained personnel for technical jobs and the deviation of young men into military training will accelerate the rise in the average age of industrial workers. War would further speed this rise. Problems of placement, retirement, the utilization and conservation of the health of older men in key positions of great responsibility, the complexities of workmen's compensation laws in relation to occupational exacerbation of pre-existent disease and many more questions are becoming increasingly urgent. Certain problems of adult education have already been mentioned. Problems involving social attitudes toward the aged are distinctly pertinent.

These three major divisions of the problems of gerontology are intimately inter-related. Advance in any one field depends on parallel or preceding advance in the other categories. It cannot be overemphasized that the more we know about the fundamental biologic mechanisms of aging, the more effectively can clinical medicine treat and understand the aged and aging. Likewise, the greater the clinical knowledge concerning the changing capacities and limitations of older people, the more intelligently can the serious and complex socioeconomic problems be considered. Knowledge, gained by painstaking, tireless and honest research, is the foundation of progress.

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The question arises whether preventive geriatrics is a public-health problem at all. If guardianship of the health of the nation is the function of public-health services, all preventive medicine logically falls within the scope of our responsibilities. As previously suggested, preventive medicine is of two types. These may be called the impersonal or "wholesale" type and the individual or "retail" form of prophylaxis.

Impersonal public-health activities have included the institution and control of modern sanitation, the enactment and enforcement of both local and national quarantine regulations, the control of avoidable hazards of atmospheric contamination and of the physical environment in industry and in schools, public buildings and homes, educational activities in schools and elsewhere directed toward the control of communicable diseases and, last, but not least, the encouragement and application of mass immunization against certain infective diseases. These methods have resulted in magnificent improvement in the health of our youth. However, it is notable that although life expectancy at birth increased fourteen years in the quarter of a century from 1910 to 1935, life expectancy at fifty years increased less than three years. There is reason to pause and consider why the gains have been so asymmetric.

Personal preventive medicine, on the other hand, requires individualized handling of health prob-

lems. Such health-maintenance activities are illustrated by the better student-health services and periodic examinations in industry and to a more limited degree by practitioners of medicine. Included in programs of individual health maintenance must be provision for therapy to control in their incipiency the progressive disorders of later life. There are four types of therapy, but we have descriptive adjectives for only three of them: prophylactic therapy, curative therapy and palliative therapy. The fourth is that form of treatment intended to control noncurable disorders and retard their progression. We might temporarily call it "controlative" or "retardive" therapy for want of a better term.

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Both these approaches toward health maintenance have their limitations. Impersonal preventive medicine, which has been the mainstay of public-health work in the past, is limited in its effectiveness almost solely to infective and communicable diseases. It is applicable only to groups with a community of interest. It is feasible only with relatively homogenous groups in which a minimum of individualization is requisite. Mass production is effective only so long as the units dealt with are identical or nearly so. With increasing age, individual variability increases. Individuality is a composite of intrinsic and inherited characteristics as modified by the accumulated experiences and vicissitudes of existence. As we age, we each acquire a highly personal set of experiences, mental actions, reactions, infections and intoxications. The greater the age, the greater the variability between individuals.

The limitations of personal preventive medicine as applied to older persons are quite different. In the first place, a great deal of time is required for proper individualization. The early detection of chronic and progressive disorders is more difficult and time consuming than the diagnosis of frank acute disease. The disorders met with in older people are usually asymptomatic in their incipiency, when most can be accomplished therapeutically. Frequently, extensive diagnostic and functional studies are necessary to reveal occult defects, which are, nevertheless, significant, for they are often precursors to extensive impairment of health. Furthermore, to prevent, control or retard these disorders, frequent consultations for observation and guidance are necessary. Such work cannot be hurried and requires the highest type of diagnostic acumen. Thus, the practice of individual prophylactic geriatrics is of necessity relatively expensive.

Another and severe limitation to the effectiveness of individual preventive medicine lies in mankind's curious perversity in declining to make prophylactic efforts on his own behalf. The best advice is relatively useless if not followed. Impersonal preventive medicine deals largely with matters that require little or no effort on the part of the recipient of the benefits. The city dweller has his sanitary milk and water, his clean food and his protection against communicable diseases without directly participating in the work necessary to ensure these blessings. Personal efforts, often of considerable degree and sometimes involving irksome restrictions, are absolutely essential in the management of early degenerative disorders to retard their progression. Unlike the majority of infectious and acute diseases, chronic disease is not infrequently etiologically associated with indulgence. This is particularly notable in diabetes mellitus, obesity and certain forms of vascular disease.

Another difficulty arises from the fact that although new curative methods of treatment are immediately taken up by both physicians and the lay public, prevention of disease is not regarded as a matter of urgency, and adoption is therefore slow. Geriatric disorders, being insidious and painless in their beginning, are usually neglected; those diseases that hurt early receive prompt attention. With chronic progressive disorders, the patient usually waits until the damage is irreparable, and then expects, nay even demands, that the physician casually shake a miracle from his sleeve. This occurs with such tragic frequency that the physician begins to wonder if pampering paternalism has not erased from the public consciousness the recognition that responsibility for health rests first and foremost on the individual.

Prevention lacks the drama of cure. It is axiomatic that volunteered or free advice is almost never followed. Advice must be sought to be used, and should be paid for if one expects conscientious application. Price as a criterion of value is a misconception that cannot be eradicated in less than several generations. It is extremely difficult to sell prophylaxis. The benefits are revealed only statistically. Statistics are abstractions and thus have very little appeal to the emotions of egocentric mankind. One cannot say, "Do not cross railroad tracks without looking, for you will surely be hit by a train the first time you try it." This is not true. One can say, however, "You will probably live to cross more railroad tracks if you always stop, look and listen. Those who do not, take chances." But those who prefer to take chances, to gamble rather than make an effort, are in the majority.

The greatest obstacle to effective preventive geri-

atrics is this inertia. It is not, however, an insurmountable obstruction. Vaccination was introduced against violent agitation. Even today there is much passive and some active resistance to many procedures of preventive medicine. Education is gradually diminishing this resistance, and has often caused parents to seek prophylactic immunization for their children. But education is not accomplished overnight. The course in education to overcome the general inertia of adults toward personal preventive medicine will be a long and arduous program. The deeply ingrained tendency to hear only what we want to hear and see only what we want to see must be combated continuously. And such education must start with the physician, not only as teacher but as student as well. Physicians are proverbially neglectful of their own health and notoriously unmindful of the potentialities of prophylaxis. Let it never be forgotten that example is the first principle of successful pedagogy!

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With the indication why impersonal or community approaches to health maintenance are inadequate to stem the rising tide of disability due to chronic disease among aging adults, the question arises what can be done to facilitate the application of individual prophylactic medicine or preventive geriatrics. Education is the primary step. It is essential that every opportunity be grasped to reiterate and emphasize that health is more than the mere absence of disease. Health has quantitative attributes. Few, indeed, are those who are truly and fully healthy. Physical and mental fitness are not static. Health involves reserves of functional capacities in excess of daily requirements and adaptability to shifting burdens.

The measurement of physical health is fraught with difficulty. Thus far, no one has formulated an adequate minimum routine of examination to reveal the degree of health. Unquestionably, many of our criteria of normality are unsuitable, for there has been but little consideration of age as a variable. The changes of age affect the criteria of health. Arbitrary standards based on chronologic age do not suffice, for biologic age is not necessarily the same as chronologic age. The greater the duration of life, the wider will be the fluctuations between physiologic and chronologic age. The question of how physiologic age may be measured is perhaps the most urgent of all the clinical problems of gerontology. The physician applies that indefinable something we call clinical judgment and arrives at an approximate estimate. There is little precision in such evaluations, however. A logical approach to quantitation of biologic age is through the measurement of several functional reserve capacities. Study of several is necessary, for aging does not proceed uniformly throughout the individual. Depletion of reserves occurs long before actual functional failure, when subjective symptoms arise. Such depreciation of reserves is detectable only by the application of controlled stress procedures eliciting responses to increased physiologic work as part of the health inventory. For example, it takes a steep climb to reveal the weak spots of an automobile engine or of an apparently healthy heart. No single observation of normal findings on physical examination can reveal how the body will react under conditions of stress. The pulse rate may be normal at rest, but may rise to excessive heights on effort; the blood pressure may be within normal limits during calm conversation, but may soar to dangerous levels with anger or fear.

Periodic health inventory is the foundation for personal preventive medicine. The term "inventory" is used advisedly, for it is far more comprehensive than the more commonly employed term "examination." The average layman expects an examination to be completed in about half an hour. An adequate health inventory requires much, much longer. A careful, painstakingly detailed history is important. The details of the past are of accumulative value to the physician. Personality plays a major role in personal hygiene for health maintenance. Information gained from laboratory procedures, x-ray studies and functional tests is accumulated and correlated with the other observations. Then, and only then, can the physician evaluate the degree of health of an individual patient. These data make possible the formulation of sound and considered recommendations. It is the prophylactic advice that determines the value of periodic health audits. It is of little profit to the patient if the physician merely records his observations on the card and does not amplify his remarks. Because of this and also because many such inventories are carelessly and superficially conducted, the periodic examination has fallen into disrepute in many quarters. There is no weakness in the principle, but the methods of application have frequently been woefully poor. All too often, the doctor remarks that the patient appears a little "run down" and prescribes a "tonic." This may be equivalent to whipping or spurring a tired horse. To be sure, it makes him go faster for a time, but it hardly helps the horse. The only effective remedy for fatigue is rest.

Rest, to be effective, requires time. Time is a vitally important element to all those concerned with geriatrics. In the first place, time is obviously a factor in aging per se. Secondly, repara-

tive processes are slow, and much more time is required to accomplish the same degree of physical rehabilitation in older persons than in children. Clinical experience shows that a man of fifty may require ten times as long to recuperate fully from an acute infection as a child of five. Therefore, convalescence must be prolonged in proportion to age. The human body has a liberal credit structure on which it may draw in periods of emergency. Violent exertion may lead to an oxygen deficit, which may require some little time for repayment when hyperpnea is continued after the cessation of exertion. Likewise, one may "borrow" against the future by "running on one's nerve," by using stimulating drugs, such as caffeine, or by ignoring the warnings of fatigue. Such borrowing is against the tissue reserves of energy and fuel. Sooner or later, these debts must always be paid. With aging, credit gets tighter. Not only is there less margin, but the call for payment becomes more urgent. Fatigue comes on more readily, and more quickly turns into exhaustion. It is increasingly imperative, therefore, that the warnings of fatigue be heeded as we grow older. Preventive geriatric advice should include emphasis on the importance of slowing the pace, avoiding overfatigue and taking sufficient time for recuperation. Thus, the final reckoning may be postponed.

Adequate prophylactic counsel must include many aspects of the patient's life and daily hygiene. The mode of living, adequacy of sleep, character of work, forms and amount of exercise, extent of relaxation and investment of leisure all play parts in the hygiene of health. Careful consideration of the habitual dietary is essential. With older people, the questions of bulk, vitamin intake and fluid balance are particularly pertinent. It is curious how often water is forgotten in considering dietary imbalances. Psychic factors must not be ignored. Many unnecessary anxieties are associated with both the masculine and feminine climacteric, and many avoidable psychic traumas from family attitudes toward the aged may be prevented by timely explanation. Conflicts due to competition with youth at work are frequently benefited by simple airing.

Aging accentuates idiosyncrasies, and certain fads may become ingrained convictions. So long as these dietary or other fads do no harm, it is wisest to leave them alone. The aged person knows quite well what he or she cannot tolerate in the way of specific food aversions. The keynote of all advice to the healthy aging or aged patient is: moderation in all things — in work, play, exertion, food and drink. Much mischief may be done by sudden and radical curtailment of such habits as the use of tobacco, alcohol or coffee.

If a careful health inventory reveals the beginnings of disease, active therapy should be instituted promptly. As previously mentioned, it is a characteristic of the diseases of late maturity that they are progressive. The earlier retardive or controlative management is applied and the more continuous is the guidance, the better are the results. Although delay in instituting proper therapy on the discovery of an early case of diabetes or hypertensive arterial disease is less likely to have tragic consequences than the neglect of an early case of cancer, this fact never justifies postponement of therapeutic measures. Procrastination permits the degenerative changes to progress to the point where treatment becomes ineffective. It is beyond the scope of the present discussion to consider the principles of active treatment in geriatric medicine.

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There is much that public-health services can do to forward preventive geriatrics. Education of the adult population in the importance of prevention, control and retardation of the chronic and progressively disabling diseases of later life will encourage the seeking of such periodic inventories as those just described. If people request these and are aware of the necessity for comprehensive thoroughness, the services will become available. Today, it is often extremely difficult, even in the larger cities, to find physicians whose primary interest is to maintain health rather than treat disease. Pediatricians are the exception. Yet it is clear that such personal preventive medicine is necessary if we hope to check the ever-increasing numbers of middle-aged and elderly people partially or totally disabled by chronic illness. The economic burden of chronic illness is already a staggering fraction of the total losses due to ill health. Mental disability requiring institutional care must be included in these considerations. It is noteworthy that admissions to state hospitals for mental disease due to cerebral arteriosclerosis jumped in the last twenty-five years from 7.7 to 49 per 100,000 population over forty years of age; this is an increase of over 500 per cent.

Industry is much concerned with these questions, and industrial medicine is in a particularly advantageous position to advance preventive geriatrics. To emphasize the value of adult preventive medicine, it is necessary only to mention that sickness absenteeism of nonindustrial origin is responsible for about twenty times as much lost time as industrial injuries of all sorts. Not only do industrial physicians have the oppor-

tunity to examine hundreds of thousands of new employees each year but also they have the invaluable privilege of examining these same men and women at periodic intervals for many years. The factory stands in much the same relation to many adults as the school holds in relation to children. The role of the school physician and school nurse in developing prophylactic pediatrics was and is most significant. A similar opportunity to develop and apply prophylactic geriatrics awaits industrial physicians and nurses. Divisions of industrial hygiene in health departments can do much to encourage the expansion of such activities. Prophylactic geriatrics does not start at any particular age; health in maturity is good insurance toward healthy old age.

There remains for brief discussion the greatest and most important service that public health can offer the older fraction of the population: research into the problems of aging. Here the field is so wide, so untrammeled and so unexplored that the potentialities are truly without end. I have deplored our ignorance of the fundamental biologic process of senescence. Badly needed are accurate and controlled studies to reveal what actually happens with aging. Cytologic, biochemical and physiologic definition of the changes that are part of normal aging are necessary as base lines on which to place evaluation of what constitutes "normal" in relation to age. The relations of age to growth and growth potentials, the changing vulnerability of cells to noxa and to pharmacologically active substances urgently require illumination. Nutritional requirements and tissue respiration in relation to age are fertile fields for study, and are ready for cultivation. The etiology and pathogenesis of arteriosclerosis, hypertensive arterial disease and cancer have obvious but ill-defined relations to the aging process.

Clinical researches are needed just as urgently. Only a few of the many unanswered questions clamoring for solution are the problems of how to evaluate physiologic age in man, and the definition of the curves of "normal" in relation to age. Such knowledge is prerequisite to studies of most geriatric diseases. Present clinical methods of functional mensuration need critical appraisal, and new, more precisely controlled, stress-test procedures are wanted. Any advance in knowledge concerning the so-called "degenerative diseases" advances geriatrics, but we must not forget the lesson learned from pediatrics, namely, that the greatest advances were made when the peculiar structural, biochemical and physiologic characteristics of the infant and child became more clearly defined.

The socioeconomic problems of the aging population are not beyond the ken of public health. The problems of industrial health, housing, optimum nutrition and old-age assistance and the costs of chronic illness, chronic-disease hospitals and the maintaining of mental health are all borderline questions.

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In brief recapitulation, it should be emphasized that the three major categories of problems, the biology of senescence, the clinical questions of aging man and the socioeconomic problems, are all intimately related. Advance in one field will be immediately reflected by progress in the others. The more we know concerning the fundamental processes of aging, the more effective can clinical geriatrics become; the more precisely clinical medicine can define the changes in capacities and limitation that occur with normal aging and the more effectively it can apply preventive geriatrics, the more intelligently can the socioeconomic problems be attacked. These three divisions cannot be arbitrarily separated. Comprehensive programs of research must consider the field as a whole.

Prophylactic geriatrics does not take as its primary objective the prolongation of life. Rather, in the words of George Morris Piersol, does it aim to "add more life to the years rather than more years to life." By understanding and developing the capacities of older persons, it can help them to remain useful much longer. There is no greater tragedy for the aged than the sense of uselessness that society today prematurely imposes on them.

It must not be forgotten that the posts of greatest responsibility, requiring the highest judgment, technical training and wisdom, are held by older men. And with good reason. The accumulated judgment and wisdom of these older minds represent one of the most valuable and potent resources of the nation. The conservation of the health and vigor of these almost irreplaceable older men is a major potentiality of preventive geriatrics.

CERTAIN OBSERVATIONS IN LOW-NITROGEN, NORMAL-OXYGEN ATMOSPHERES RELATED TO THE PROBLEMS OF HIGH-ALTITUDE FLYING*

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IN the course of studying the effect of low-nitrogen, normal-oxygen atmospheres on the dyspnea of obstructed breathing,[1] observations were made on the effects produced by such atmospheres that bear directly on the problems of high-altitude flying. Because these effects have become of particular importance in current aerial warfare and in low atmospheric-pressure experiments pertaining thereto, publication of our findings seems desirable.

The experiments were conducted in a steel chamber,‖ which consisted of two rooms. In each room, controls by suitable pipes and valves were made so that from either inside or outside the pressure chamber the room could be evacuated rapidly and almost noiselessly by means of a steam ejector to any desired extent and could be refilled with room air or oxygen as desired. A bank of oxygen tanks was provided with a reduction valve so that an adequate source of oxygen at a pressure of 50 to 100 pounds was available. Cooling of the chamber was effected by a coil carrying brine. The accumulation of carbon dioxide was prevented by frequent small reductions of total pressure and additions of oxygen and air. Convection currents facilitated the absorption of carbon dioxide by a container of soda lime. Water pipes to each chamber provided the essential fire-extinguishing equipment.

By repeating the process of alternately lowering the total atmospheric pressure until the partial pressure of oxygen was reduced to approximately 125 mm. of mercury and then increasing the partial pressure of oxygen to approximately 225 mm., a total atmospheric pressure of 200 mm. (corresponding to an altitude of approximately 32,000 feet) and a partial oxygen pressure of 155 mm. could be provided within the chamber in ten minutes. A fairly accurate account of the oxygen pressure was kept by calculation from the pressure changes. An accurate check was made at intervals by direct oxygen analysis, which took but two or three minutes.

From observations during experiments at low-nitrogen, normal-oxygen atmospheres, Mosso,[2] Winterstein,[3] Fegler[4] and Dill, Edwards and Robinson[5] postulated an excess carbon dioxide elimination that was not dependent on the hyperpnea of anoxemia. Ruhl[6] found that the slight lowering of the carbonic acid of arterial blood in such atmospheres was accompanied by a compensatory decrease in bicarbonate and no change in the pH of the blood. Armstrong[7] in similar atmospheres observed no abnormal elimination of carbon dioxide.

Table 1 presents our observed and calculated[1] alveolar oxygen pressures at total atmospheric pressures corresponding to varying altitudes, when the atmospheric oxygen pressure was maintained at the sea-level oxygen pressure of 155 mm. of mercury. The relative constancy of the alveolar car-

TABLE 1. *Alveolar Oxygen and Carbon Dioxide Pressures and Percentage Saturation of Hemoglobin at Atmospheric Pressures Corresponding to Various Altitudes When the Atmospheric Oxygen Pressure Is Maintained at the Sea-level Value.*

Altitude	Atmospheric Pressure		Calculated* Alveolar Oxygen Pressure	Observed Alveolar Oxygen Pressure	Observed Alveolar Carbon Dioxide Pressure	Calculated† Arterial Hemoglobin Saturation
	total	oxygen				
ft.	*mm. Hg.*	*mm. Hg.*	*mm. Hg.*	*mm. Hg.*	*mm. Hg.*	%
0	759	159	104	100	40	98
6,000	600	150	94	105	40	96
11,000	504	156	96	112	39	97
17,000	400	156	93	100	37	95
19,000	360	158	93	87	40	95
23,500	300	156	87	84	37	94
27,700	250	156	81	79	39	93
32,500	200	159	77			92
37,500	159	159	67			90
40,000	140	140	48			80

*Assuming a constant alveolar vapor pressure of 48 mm. of mercury, a carbon dioxide pressure of 40 mm., a respiratory quotient of 0.9 and no hyperpnea:

$$\text{Alveolar oxygen pressure} = (\text{Atmospheric pressure} - \text{water-vapor pressure}) \times \frac{\text{partial oxygen pressure}}{\text{atmospheric pressure}} - (\text{alveolar carbon dioxide pressure} \times \frac{1}{0.9}).$$

†Determined from the calculated alveolar oxygen pressure.

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‖The plan of the chamber was conceived by Dr. James L. Wilson. Its construction was supervised by Dr. Wilson and Professor Philip Drinker, of the Harvard School of Public Health.

bon dioxide pressures over this range in altitude does not indicate an abnormal elimination of carbon dioxide. That there was no significant alkalosis due to abnormal loss of carbonic acid from the blood is indicated by the fact that there was no increase in urine volume or alkalinity such as would accompany a respiratory alkalosis.[8] Thus our results do not indicate that in rarefied atmospheres there is a significantly increased elimination of carbon dioxide that is independent of the hyperpnea of anoxemia.

Fegler[4] and Dill and his co-workers[5] believed that the greater elimination of carbon dioxide observed by them at low pressures resulted from the greater diffusion of carbon dioxide from the alveolar spaces to the pulmonary dead space when the air in the lungs was reduced in density. It has

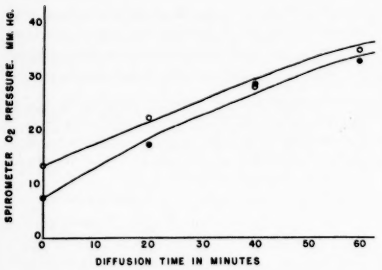

FIGURE 1.

This chart shows the rates of diffusion of oxygen into a spirometer at a constant partial oxygen pressure of 150 mm. of mercury at total atmospheric pressures of 760 mm. of mercury (open circles) and of 300 mm. of mercury (solid circles).

similarly been suggested[9] that an acceleration of the diffusion rate of oxygen at low-nitrogen pressures facilitates respiration. To obtain data on the diffusion of oxygen at atmospheres of varying density, a large spirometer was filled with nitrogen after the oxygen of the dead air space of the apparatus had been washed out with nitrogen. The spirometer was then opened to the surrounding atmosphere by removing the two corks at its base, thus allowing two apertures each about 2 cm. in diameter. At the beginning of each experiment and at intervals thereafter, samples were drawn off for quantitative analysis of oxygen content. Such experiments were conducted at sea-level pressure with room air and at 300 mm. of mercury total pressure, with a partial pressure of oxygen of 150 mm. Figure 1 gives the partial pressure of oxygen plotted against the diffusion time in minutes. The curves show a similar rate of diffusion at the two atmospheric pressures. This suggests that acceleration of the diffusion rate of oxygen does not significantly facilitate respiration at low-nitrogen pressures.

Bert,[9] Boycott and Haldane[10] and Haldane[11] called attention to the theoretical possibility of "bends" if the atmospheric pressure was reduced too rapidly from that of sea level to 300 mm. of mercury or less. Von Schrötter[12] recorded symptoms resembling "bends" after reduction of the total pressure in a tank to 230 mm. in fifteen minutes but believed the symptoms could not have been "bends." In experiments in which the pressure was reduced rapidly to 250 mm. or less, we observed on several occasions one or more of the following symptoms: joint, muscle, precordial and thoracic pains, rapidly spreading skin anesthesia and scotoma. Since then, Armstrong[7] has reported similar experiences. At pressures of 250 mm. of mercury or less, we regularly observed a burning sensation of the eyes and tingling and other paresthesias of the skin. At such pressures, we were also aware of an increase in the effort required for physical and mental work. Inertia prevented a ready response to any task. Armstrong has likened this to the symptoms of anoxia without anoxemia. These symptoms observed independently by Armstrong and ourselves and Armstrong's evidence[7] concerning the formation of nitrogen bubbles and the occurrence of increased intracranial pressure indicate that the possibility of decreased-pressure disease, that is, "bends" and increased vaporization of body water, is a real danger. Although preparatory elimination of nitrogen by breathing oxygen prior to ascent lessens the danger of "bends," this precautionary procedure will not remove the danger that may accompany the increased vaporization of body water at low atmospheric pressures.

Armstrong and Heim[13] have given an excellent description of the adjustment of pressure within and without the middle ear during ascent and descent. In ascent, the expanding air in the tympanic cavity automatically escapes through the eustachian tube. In descent, however, the increasing external pressure closes the eustachian tube, and equilibrium can be established only by opening the tubes by voluntary contraction of their dilator muscles. If the differential pressure becomes greater than 90 mm. of mercury, the muscles can no longer open the closed tubes, and the ready establishment of equilibrium becomes impossible. A differential pressure of 30 mm. causes pain, and an increase in the differential external pressure of 100 mm. or more usually results in rupture of the eardrum. In our experi-

ence, the readiness with which closing of the eustachian tubes accompanied rapid descent varied greatly with the subject, and was also dependent on whether or not congestion and inflammation of the mucous membrane of the nasopharynx were present. Certain subjects encountered great difficulty in preventing closure of the tubes by the usual procedure of swallowing and yawning, even during relatively slow descent, whereas others encountered no difficulty with very rapid descent.

Preliminary observations on the burning of matches and cigarettes revealed a marked increase in their rates of combustion at low-nitrogen, normal-oxygen pressures. With the aid of Professor Philip Drinker, a controlled series of experiments on the rate of burning of carefully calibrated candles was carried out in atmospheres of different nitrogen pressures but with a constant partial pressure of oxygen of 150 mm. of mercury. The results are plotted in Figure 2. This increased rate of combustion at the low-nitrogen pressures introduces a fire hazard that deserves consideration and seems to have been overlooked by other workers. Our data suggest that pressure chambers for low-pressure experiments or pressure-

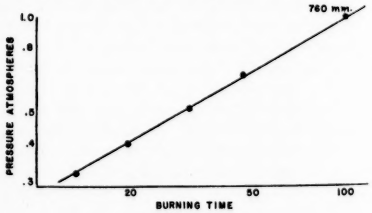

FIGURE 2.

This chart shows the increase in rate of combustion or decrease in burning time with a decrease in total atmospheric pressure but a constant partial-oxygen pressure of 150 mm. of mercury. The scales of ordinate and abscissa are logarithmic.

cabin planes for high-altitude flying should be equipped with adequate fire-extinguishing equipment that will withstand the pressure changes and will not liberate toxic gases.

Finally, the low values for alveolar oxygen observed at total pressures of 250 mm. of mercury or less, in spite of the adequate partial pressures of oxygen, demonstrate a limit to high-altitude flying unless the total atmospheric pressure, as well as the partial pressure of oxygen, is maintained above certain limits. In Table 1, the calculated alveolar oxygen and arterial hemoglobin saturation have been extended to total pressures below, and thus to altitudes above, the levels at which the alveolar air was actually determined. The risk involved in handling mercury in a closed chamber at markedly reduced pressures, owing to the marked increase in vaporization, was one of the factors that limited our analytical experiments. The data demonstrate that at altitudes above 32,000 feet or at total pressures below 200 mm. of mercury the alveolar oxygen and arterial hemoglobin saturation fall off significantly and with increasing rapidity, in spite of a partial-oxygen pressure of 159 mm. This is accounted for by the normal alveolar carbon dioxide and water-vapor pressures of 40 and 48 mm., respectively. At a total pressure of 140 mm., the sum of these normal alveolar carbon dioxide and water-vapor pressures permits, even if pure oxygen is breathed and the respiratory quotient is 1.0, an alveolar oxygen pressure of but 140 mm. minus 88 mm., or 52 mm., of mercury unless hyperpnea is present. This pressure is not very much above the normal venous oxygen pressure of 40 mm. of mercury. Hyperpnea increases the alveolar oxygen pressure, but unless the atmospheric carbon dioxide pressure is increased, results in decreased alveolar carbon dioxide pressure and alkalosis. An atmosphere of pure oxygen at a total pressure of 130 mm. of mercury or an altitude of 42,000 feet would probably result not only in the same potential dangers due to anoxemia as are encountered in ordinary air at 22,000 feet but also in the dangers due to reduced-pressure disease. Indeed, a recent report by Ceres[14] suggests that effects of the latter are encountered in flights above 30,000 feet.

Thus, to reach altitudes greater than 32,000 feet with any degree of safety, provision must be made for controlling not only the partial pressures of oxygen but also the total atmospheric pressure. This requirement, the middle-ear disturbances in descent, the possibility of "bends" in ascent and of increased vaporization of body fluids at very high altitudes, and the fire hazard at low-nitrogen, normal-oxygen pressures result in great vulnerability at altitudes much beyond 35,000 feet.

SUMMARY

Observations related to problems of high-altitude flying made during experiments at atmospheric pressures of 250 to 200 mm. of mercury, corresponding to altitudes between 28,000 and 32,000 feet, are presented and discussed. The increased rate of combustion observed in low-nitrogen, normal-oxygen atmospheres is such as to cause a fire hazard that deserves careful consideration and precautions.

We are indebted to Professor Philip Drinker, Mr. Charles Miller and Miss Elsie MacLachlan for their co-operation and assistance in the course of these experiments.

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CHRONIC GASTRITIS: A GASTROSCOPIC AND CLINICAL STUDY*

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THIS communication reports the results of studies of chronic gastritis in adults, the diagnosis of which was made by means of gastroscopy. There were three purposes for making these studies. The first was an investigation of the symptomatology, including an analysis to ascertain whether certain symptoms would permit a presumptive diagnosis of chronic gastritis before performing gastroscopy. The second purpose was to collect statistics on the incidence of chronic gastritis, as observed gastroscopically. A third was an attempt to discover whether the existence of chronic gastritis in the presence of peptic ulcer influenced the symptomatology of the latter condition.

Schindler[1] has developed a practical classification of the main types of chronic gastritis based on gastroscopic studies. An elaboration of this classification, suggested by McClure and Jankelson,[2] embraces the various types of chronic gastritis observed in over 1000 gastroscopic examinations. This classification comprises three main types: the superficial, the atrophic and the hypertrophic, each of which may occur as the simple, ulcerating or hemorrhagic form. Except for the superficial, these terms are self-explanatory. The superficial type is a chronic gastritis in which the mucosa appears inflamed, with redness, swelling and edema and an excess of mucus. The above classification will be followed in describing our observations.

The pathologic characteristics of various types of gastritis were first established by the postmortem studies of Faber.[3] His findings were subsequently confirmed by the extensive observations of Konjetzny[4] on gastric mucosa resected during laparotomy, and more recently by Eusterman,[5] Schindler[6] and others. However, the practical clinical diagnosis of gastritis was not possible until the development of the Wolf-Schindler semiflexible gastroscope in 1932. This instrument permits the direct visualization of mucosal changes such as the various types of chronic gastritis, erosions, superficial ulcerations, purpuric manifestations and other lesions of the gastric mucosa not otherwise readily demonstrable during life. As a result of the use of this gastroscope, a large literature on gastroscopic findings has accumulated and has been correlated by Schindler,[1] Swalm, Jackson and Morrison,[7] Swalm and Morrison,[8] Schindler and Murphy[9] and in reviews by Cheney,[10] by Jones et al.,[11] and by others. All the observers agree that chronic gastritis is a common gastroscopic finding. This frequency is well illustrated by Schindler's[12] tabulation of the gastroscopic findings in 1000 patients, in whom the lesion was present in 42 per cent; Carey[13] found it in 44 per cent of 700 gastroscopies. Nevertheless, more recently Ruffin, Brown and Clark[14] conclude from the results of their gastroscopic observations that the reported frequency of chronic nonspecific gastritis is to be seriously doubted.

Most of the reported gastroscopic studies have been largely concerned with descriptions of the gross anatomic changes observed, and but incidentally with the accompanying symptomatology. How little differential diagnostic knowledge has been accumulated is illustrated by a quotation from Swalm et al.,[7] "The symptoms of gastritis are so variable that they range from a complete absence of symptoms to flatulent dyspepsia to perfect mimicking of peptic ulcer with its hunger pains and food or alkali relief with hematemesis." Nevertheless, more recent studies[9, 15–17] suggest the possibility of clarifying the symptomatology. These studies indicated that in patients with gastritis the appetite was often poor, although it might remain unaffected but was not found to be increased. Relief after food was found to be less common than in ulcer; indeed, food not infrequently aggravated the symptoms. Epigastric distress or true pain was the rule and was often of the delayed type. Severe epigastric pain, not referred, was reported only in the hypertrophic form.[15] Hemorrhage was an important complication.[1, 17–20] It was suggested[9] that the atrophic type may be differentiated by the general symptoms of marked weakness, sense of fatigue, emotional depression, sore tongue and paresthesias. Although the degree of gastric acidity was reported[9, 15, 21] to be variable, achlorhydria or hypoacidity was more commonly found in the atrophic type and normal or increased values in the superficial and hypertrophic forms.

The incidence of any pathologic condition is always of great importance. For this reason, we determined the frequency of chronic gastritis by means of gastroscopic observations in 611 consecutive patients; it was present in 269, an incidence of

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44 per cent. Although 99 (37 per cent) of these patients showed mixtures of the different types of gastritis, in each of them one type predominated sufficiently to permit a classification as essentially hypertrophic, atrophic or superficial. On this basis, 150 (56 per cent) were of the hypertrophic, 51 (19 per cent) of the atrophic, and 68 (25 per cent) of the superficial type, an incidence of 25 per cent, 8 per cent and 11 per cent, respectively. The findings are comparable with those reported by other observers,[11, 14] and show that various types of chronic nonspecific gastritis are of common occurrence. The associations of the types of gastritis mentioned above were hypertrophic with superficial in 33 cases, hypertrophic with atrophic in 29 cases, atrophic with superficial in 25 cases, hypertrophic with superficial and atrophic in 4 cases, and ulcerating or hemorrhagic ulcerating with either hypertrophic or atrophic in 8 cases. These figures represent incidences of 5.4, 4.7, 4.1, 0.7 and 1.4 per cent, respectively.

For the purpose of evaluating the symptomatology of chronic gastritis, 75 patients were subjected to comprehensive studies. Obviously, the thoroughness of such studies is an essential factor governing their value. For this reason, with certain exceptions, all observations and procedures incident to the clinical study were carried out with the utmost care by at least one of us; and in every case gastroscopic examination was made by two of us. The exceptions mentioned refer to the radiographic and laboratory procedures, the majority of which were performed in the appropriate departments of the hospital.

The clinical studies showed that a small number of patients were troubled with constipation, but in most of them the bowel habits were not unusual. Weight loss was sustained by 6 patients with hypertrophic and by 4 with atrophic gastritis. The loss was approximately at the rate of 1 pound a week and was never extreme. Twenty-nine of the patients with hypertrophic and 25 with atrophic gastritis showed no weight loss. Thus, changes in weight in this series of patients were not of clinical importance. Except as the result of gross hemorrhage, the blood and stools showed nothing demonstrably abnormal. The Hinton test on the blood serum was negative in all the patients, as was the usual clinical examination of the urine.

Thirty-nine of the patients presented hypertrophic gastritis, and 29 atrophic gastritis. Of the former, 34 were men and 5 women, whereas of the atrophic, 17 were women and 12 men. Thus, men predominated greatly in the hypertrophic and women to a lesser extent in the atrophic type. Table 1 shows that the ages varied from twenty to eighty years, with a rather even distribution of the hypertrophic cases for each decade; the atrophic type showed lessened incidence in the sixth and eighth decades. However, in general the age incidences found for the two types of gastritis are comparable.

TABLE 1. *Ages of Patients.*

AGE	NUMBER OF PATIENTS: HYPERTROPHIC GASTRITIS	NUMBER OF PATIENTS: ATROPHIC GASTRITIS
yr.		
20 to 30	6	7
30 to 40	5	7
40 to 50	8	5
50 to 60	8	2
60 to 70	10	7
70 to 80	7	1

The duration of the symptoms for both types of gastritis is outlined in Table 2. In general, the duration was comparable in each group. This tabulation also indicates that gastritis is a chronic condition.

Of the 68 patients showing hypertrophic or atrophic gastritis on gastroscopy, 20 had a duodenal ulcer and 1 a gastric ulcer, as diagnosed by x-ray studies, and 6 presented x-ray evidence of gall-bladder disease or had undergone cholecystectomy. The presence of the gastric ulcer was verified by gastroscopic observation. The remaining 41 cases were uncomplicated; an analysis of the symptomatology of these patients is given in Table 3. Of the 41 patients with uncomplicated chronic gastritis, 30 complained of epigastric discomfort or mild pain, and 9 of severe epigastric pains. Thus, epigastric discomfort and pain were the commonest symptoms. As the table shows, they were variably related to the time of taking food, and they were also variably relieved by the ingestion of food or alkali. The peptic-ulcer characteristic of periodicity of symptoms was described by 24 of the patients. Good appetite was present in 32, and anorexia in 12. Vomiting was a prominent symptom in 15, and hematemesis in 5. Of the 2 patients with atrophic gastritis without pain

TABLE 2. *Duration of Symptoms.*

DURATION	NUMBER OF PATIENTS: HYPERTROPHIC GASTRITIS	NUMBER OF PATIENTS: ATROPHIC GASTRITIS
Less than 1 yr.	4	8
1 to 5 yr.	16	10
5 to 10 yr.	3	6
10 to 15 yr.	6	1
15 to 20 yr.	3	1
20 to 25 yr.	7	1
25 to 40 yr.	0	0
40 to 45 yr.	0	2

or discomfort, one complained of nausea and fatigue and the other complained of fatigue and also of the regurgitation of small amounts of bloody liquid. Fatigability occurred in 33 per cent of the atrophic, but was absent in the hypertrophic, group.

The data correlated in the table are the results of careful scrutiny of each patient's history. They indicate the basis for concluding that, except for fatigability and absence of epigastric complaint in 2 patients of the atrophic group, the symptomatology of hypertrophic and atrophic gastritis is similar. The case scrutiny also disclosed that 10

TABLE 3. *Symptomatology of 20 Hypertrophic and 21 Atrophic Cases of Uncomplicated Chronic Gastritis.*

SYMPTOM	NUMBER OF PATIENTS	
	HYPERTROPHIC GASTRITIS	ATROPHIC GASTRITIS
Severe epigastric pain	2	7
Mild epigastric pain or discomfort	13	7
Constant or intermittent pain	10	7
No time relation of pain to meals	15	10
Pain augmented by food	6	3
Pain or discomfort relieved by food	16	11
Pain or discomfort relieved by alkali	17	9
Periodicity of symptoms, like ulcer	11	13
Symptoms typical of ulcer	5	5
Good appetite	18	14
Anorexia	7	5
Vomiting	9	6
Hematemesis	3	2
No epigastric pain or discomfort	0	2
"Nervousness"	7	9
Fatigability	0	9

patients in these two groups showed symptoms characteristic of peptic ulcer. An analysis of the symptomatology of the remaining 31 cases shows certain symptoms differing from those of the typical peptic-ulcer syndrome: no time relation of pain or discomfort to meals, 25 cases; pain or discomfort augmented by food, 9 cases; pain or discomfort not relieved by food or alkali or by both, 5 cases; constant or intermittent pain or discomfort, 17 cases; constant pain or discomfort not relieved by food and associated with sharp, knifelike epigastric pain definitely related to meals, with relief by alkali, 6 cases; anorexia, 12 cases.

Of the 21 patients showing both chronic gastritis and ulcer, modification of the usual ulcer syndrome occurred in 4. In 3, duodenal ulcer was present; one of them complained of anorexia, the second had anorexia, with no relief by food, and the third, in addition to these symptoms, stated that there was no time relation between the onset of pain and food intake. The fourth patient, who had a gastric ulcer, experienced no relief from pain after either food or alkali.

The presence of chronic gastritis caused no demonstrable influence on the symptoms presented by the 6 patients with chronic cholecystitis or by those who had undergone cholecystectomy.

In this study, x-ray observations were found to be of inconsequential value as a direct aid in the diagnosis of gastritis. However, barium gastrointestinal x-ray studies were of essential value in determining the presence or absence of esophageal lesions, cancer, ulcer of the stomach, pyloric obstruction, duodenal ulcer and lesions elsewhere in the small or large bowel. Radiography was of similar value in relation to the diagnosis of diseases of the gall bladder.

DISCUSSION

Chronic gastritis is known to be a common pathologic entity; it was observed gastroscopically in 44 per cent of our patients and in comparably high percentages by others.[12, 16] These high incidences resulted from limiting observations to those patients presenting gastric complaints. For this reason, the findings do not denote the frequency in the general population. The latter may be calculated by the use of the mean average of figures reported by the Commission on Medical Education,[22] which shows that 13.4 per cent of all patients visiting outpatient departments present digestive complaints. If gastroscopy reveals chronic gastritis in 44 per cent of these cases, the incidence among the general population is approximately 6 per cent.

The types of gastritis can be divided into primary and secondary groups.[2] The latter group includes chronic gastritis occurring in the course of either an established causal or a possibly causal pathologic condition. In our cases that were carefully studied clinically, 34 (45 per cent) accompanied peptic ulcer or gall-bladder disease. This finding confirms the conclusion drawn from a much larger number of less carefully controlled observations that the condition is a secondary entity in 50 per cent of the patients in whom it is observed. On the basis of this percentage, proper calculation shows that the incidence of primary chronic gastritis among the general population is about 3 per cent. In the aggregate, this percentage represents a large number of patients. Furthermore, the results of the present investigation and those of other observers[15–17, 23] show that it is a chronic disturbance, causing mild to serious and even incapacitating disability. It is also established that the condition is not infrequently mistaken for peptic ulcer, whereas it may be confused with carcinoma of the stomach,[6, 24] which has caused useless laparotomies.[24] Thus, chronic gastritis affects a sufficiently large number of patients to make its clinical investigation very important.

Morphologically, primary gastritis is indistinguishable from the secondary type, and clinical

studies furnish the only means of differentiation. Obviously, without this differentiation, the symptomatology may include a variety of manifestations due to a causative primary disease. For this reason, the analysis in this communication represents the findings only in those patients who have been intimately studied and observed for a prolonged period. This explains why the findings are reported in only 75 of nearly 400 patients in whom a gastroscopic diagnosis of chronic gastritis was made; superficial gastritis has not been discussed because the number of cases studied intensively was too limited.

A correlation of symptoms occurring in primary chronic gastritis that differ from those of the typical peptic-ulcer syndrome has already been given. These symptoms are merely suggestive, not diagnostic, of the presence of chronic gastritis. Furthermore, gastric analyses showed nothing of positive diagnostic significance regarding either the presence of gastritis or the differentiation of the two types. In addition, x-ray studies were often of inconsequential value in diagnosing the presence of gastritis. Thus, the only dependable method of diagnosing chronic gastritis or differentiating its various types is afforded by gastroscopy.

The symptomatology of chronic primary gastritis usually resembles that of peptic ulcer. However, it is occasionally suggestive of cholecystitis, and more rarely of carcinoma. In the differential diagnosis, one should consider not only these entities but also all others that may cause upper abdominal digestive symptoms, and when hematemesis has occurred, all its causes should be kept in mind. Having thus established gastroscopically the presence of gastritis, one can decide whether it is primary or secondary only by clinical means. Also, what role the gastritis plays in the production of the symptomatology can be decided only by proper clinical interpretation.

A factor useful in differential diagnosis is furnished by the findings in the 10 patients whose symptoms were typical of peptic ulcer. The differential feature is the failure to find x-ray evidence of peptic ulcer, gall-bladder disease or any other cause for the symptomatology. This is a diagnostic feature stressed by Rivers and Smith.[17] Fatigability apparently affords another diagnostic feature since, as emphasized by Schindler and his associates,[15] it is a symptom of atrophic gastritis, and since it was not observed in any case of the hypertrophic type.

Hematemesis resulting from gastritis and occurring in the absence of peptic ulcer, carcinoma, polyp, varicose veins or blood dyscrasias was observed in 3 cases of hypertrophic and 2 of atrophic gastritis; it was caused by an ulcerating type of gastritis — that is, many superficial ulcerations and fissures were scattered throughout the mucosa. The bleeding varied from mild to severe, but was not fatal.

To study the effect of chronic gastritis on the symptomatology of peptic ulcer, 21 cases of the latter disease were investigated. In only 4 of these were the symptoms apparently modified by the presence of the gastritis, the modification consisting of anorexia or absence of relief of pain by food. Thus, these symptoms, when peptic ulcer is known to exist, suggest the presence of gastritis. On the other hand, their absence does not exclude gastritis.

The tabulations already presented show that the age incidence and chronicity of the hypertrophic and atrophic types of gastritis are comparable. The findings in relation to age incidence confirm those in 21 cases of hypertrophic and 59 cases of atrophic gastritis collected from the literature.[7, 9, 23, 25, 26]

Summary

The symptomatology of gastroscopically demonstrated primary chronic gastritis was studied and analyzed. Certain symptoms were found to suggest the presence of chronic gastritis.

Hematemesis was an important complication of primary chronic gastritis.

Fatigability was a symptom characterizing primary atrophic gastritis.

The age incidence and the chronicity of hypertrophic and atrophic gastritis were comparable.

The presence of chronic gastritis can apparently be demonstrated only by means of gastroscopy. Nevertheless, the diagnostic significance of the gastroscopic findings must be governed by clinical interpretation.

The incidence of chronic gastritis among our patients was 44 per cent, and there was an approximately even division of the secondary and primary forms. It is estimated that the incidence of primary chronic gastritis among the general population is about 3 per cent.

The symptomatology of peptic ulcer was occasionally slightly modified by the presence of a demonstrable gastritis.

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MEDICAL PROGRESS

RECENT DEVELOPMENTS IN AVIATION MEDICINE*

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UNDER the stimulus of the present emergency, many laboratories in this country and abroad are investigating physiologic problems encountered in high-altitude combat flying.‡ Despite military restrictions imposed by war, the literature of aviation medicine has grown continuously, and the National Research Council has recently authorized the compilation and publication of a classified bibliography of aviation medicine, which will presently be published as a supplementary number of the *Journal of Aviation Medicine*. The following review summarizes material issued within the past two years.

The performance of modern military aircraft taxes the physiologic limitations of the pilot in many ways, and although vast sums have been spent by the Government in the design of aircraft, little so far has been appropriated for study of the physiologic limitations and protection of the pilot; this lack continues, despite the fact that in Germany some twenty independent laboratories of aviation medicine are supported by the government. The war in the air has come to be in a very real sense a physiologic war, since the limitations of air combat at the present time lie more with the pilot than with the plane, and the government that succeeds in adequately protecting the pilot through study of his physiologic needs will gain strategic advantage that will undoubtedly be decisive in determining the outcome of the conflict. It is therefore incumbent on all who are actively concerned in the present war to become familiar with the general principles of aviation medicine, especially as they relate to high-altitude combat flying.

The more important advances of the last two years relate to the following: the physiologic effects of acceleration; the use of oxygen at high altitudes; air embolism and its prevention; and anoxia of the adrenal glands. The topics are considered in this order; problems of pilot selection and training are not touched on.

PHYSIOLOGIC EFFECTS OF ACCELERATION

It is well known that modern combat flying, especially the dive-bombing maneuver, causes the development along the longitudinal axis of the body of centrifugal forces that tend to draw blood

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‡In England, the Flying Personnel Research Committee has directed research for the Royal Air Force; in Canada, a similar committee has been in session since the beginning of the war, and in the United States the National Research Council, at the request of the armed services, formed the Committee on Aviation Medicine in October, 1940. These three committees, along with the air corps which they serve, are responsible for the principal direction of research endeavor in aviation physiology in the allied countries. These committees have thought fit to publish some of their newer disclosures, but other data having strategic military significance cannot be made public at the present time, and hence a review of the subject is to this extent restricted by the exigencies of war. General principles can be discussed, but specific details of equipment must necessarily be left for the future.

away from the head, and so to induce a state of collapse from acute anoxia of the brain. The ultimate effect of a given acceleration depends on four factors: the magnitude, the duration, the direction of acceleration, and the physiologic condition of the pilot at the time the acceleration develops. An excellent discussion of the underlying physical principles is found in a recently published volume by Grow and Armstrong,[1] and in a work by Phillips and Sheard.[2] The recent German literature, with a surprising amount of experimental detail, is given by Ruff and Strughold.[3] Data from English sources are summarized by Livingston[4] and Whittingham.[5] All authorities agree that a healthy young adult can withstand a positive acceleration acting along the vertical axis of the body of 4.5 *g* (four and a half times the force of gravity) for approximately five seconds. Thereafter, vision becomes dim (the so-called "blackout"), and consciousness may be entirely lost. But from the point of view of safety in aviation, the state of the pilot's co-ordination during the seconds prior to loss of consciousness is vital, for judgment and co-ordination are poor when relative cerebral anoxia exists, irrespective of its cause. When one is flying at a high altitude under conditions of relative anoxia to start with, a small degree of acceleration may cause blackout much earlier than at sea level.

The direction of acceleration is also important, and recent studies indicate that the body withstands accelerations applied along the horizontal axis of the body far more successfully than along the vertical axis. Negative accelerations toward the head are highly dangerous because of exaggeration of the carotid-sinus reflexes. Indeed, the average young adult can seldom withstand more than 3 *g* of negative acceleration for five seconds without "seeing red" and having retinal hemorrhages. This accounts for the fact that stunt fliers are loath to indulge in any form of acceleration when flying upside down.

The condition of the organism has much to do with the capacity to withstand acceleration. All test pilots insist than an alcoholic spree of an evening considerably diminishes tolerance for positive acceleration the next day. More significant than this, however, is the fact, recently emphasized in the German literature,[3] that tolerance is seriously lowered by an empty stomach, and all German fliers are urged before going on dive-bombing maneuvers to have a full meal. The effect of food is explained by the Germans as being probably due to the filling of the visceral blood vessels—because they are filled, further blood is not readily forced into them by centrifugal action. Blood chemistry is not discussed.

In the recent German literature, the following factors, in addition to the full stomach, are mentioned as favoring the pilot's capacity to withstand acceleration:

Carbon dioxide. The inhalation of 5 or 6 per cent carbon dioxide, through augmenting the cerebral circulation, increases resistance to acceleration by 1 to 2 *g*.

Vasoconstrictor drugs. Any pharmaceutical agent that increases the tone of the capillary wall improves one's ability to withstand acceleration. Pituitrin, adrenalin and adrenocortical hormones have all been mentioned in this connection, but precise data concerning them are not available.

Pneumatic belts. Mechanical constriction of the abdomen and of the lower extremities has also been proposed in both the German and English literature to minimize the rush of blood from the head to the visceral bed and the lower extremities. Of these mechanical devices, pneumatic belts and pneumatic trousers have been the devices usually discussed. The Germans state that a pneumatic belt may increase tolerance by 1.0 to 1.5 *g*, but no one of the present belligerent countries has permitted publication of detailed reports concerning these devices.

Water suits. The Germans have also reported on a water suit designed for the prevention of blackout, and although they claim that it notably improves resistance to positive acceleration, they state that it is unsatisfactory for other reasons.*

Posture. In a recent paper by Ruff,[6] the problem of posture in relation to acceleration was discussed. The Germans, it appears, favor a crouching posture, with flexion of the legs against the abdomen, as one particularly suited for protection of the pilot against acceleration. If the pilot lies supine or prone at the end of a dive-bombing maneuver, he is also less subject to negative accelerations, but in these postures he is unable to see out or to maneuver his plane without special redesigning of the cockpit and the cockpit controls.

The problem has been studied in this country by Armstrong and Heim,[7] and the physiologic

*To quote Grow and Armstrong[1]: "The water suit is a closely fitted water-proof garment which is worn next to the skin. What little space is left in the suit after it is put on is filled with water or other suitable fluid. This causes the flier to 'float' in the suit, and during accelerations the water presses on the body equally in all directions. As a consequence the normal effects of acceleration are replaced by a uniform compression of the body which, it is estimated, could be tolerated without difficulty up to 15 *g* or more."

changes accompanying positive acceleration are admirably summarized by Poppen[5] as follows:

> Our findings confirm those of Armstrong and Heim. It is scientifically interesting that by these two methods of approach the findings were practically identical. Dr. Drinker and I found the following chain of physiological events incident to high positive accelerations: (i) A drop in carotid pressure as a result of the hydrostatic, centrifugal action on the arterial column. (ii) A drop in jugular pressure incident to the same action upon the venous column. (iii) The centrifugal effect upon the venous column is sufficient to reduce the volume of blood reaching the right auricle. This results in a reduced per-beat and per-minute output of the heart. This causes a secondary drop in carotid pressure superimposed upon direct arterial effect. (iv) The combined effects reduce the carotid systolic pressure, under moderate accelerations, from an average of 130 mm. of mercury to between 60 and 70 mm. (In one case under 6 *g*, the pressure dropped to 16 mm.) (v) The reduced pressure causes a profound slowing of the circulation through the brain. (vi) A transient local cerebral anoxemia results. This is adequate to explain the symptoms of high acceleration.
>
> We found further that the drop in carotid pressure was proportional to the acceleration. It increases on successive accelerations at frequent intervals. If the acceleration is discontinued before the secondary reduced heart output, the drop in carotid pressure is insufficient to cause severe anoxemia. This explains the statement often made by test pilots that they would prefer 10 *g* for 1.5 seconds to 5 *g* for 3 seconds.

It should be noted that all factors tending to improve the body's resistance to positive acceleration are those that tend to keep blood in the head. Hence it may be concluded that the phenomenon of blackout and loss of consciousness, which may occur within five seconds of the beginning of the acceleration, is probably due solely to acute anoxia and cannot be attributed to any direct effect of acceleration per se on the cortical neurons.

Use of Oxygen at High Altitudes

It has long been recognized that oxygen is essential for operations at high altitudes, but the reluctance of the older pilots to use it and the conservatism of the commercial air lines in supplying oxygen to passengers have tended to distract attention from the need for oxygen in the lower altitude ranges. Recently, however, there has been a change of emphasis concerning the use of oxygen; there have likewise been improvements in modes of administration of oxygen and also in supply. A general discussion of the effects of anoxia on the heart and other organs has recently been given by Hoff[9] and also by Gemmill.[10]

Need for oxygen at the lower altitude ranges. The studies of McFarland,[11, 12] Barach[12] and many observers in Europe have indicated that mental functions are gravely impaired when human beings are exposed to an oxygen partial pressure equivalent to that encountered at an altitude of 12,000 feet. Thus, when a series of Barach's students breathed 13 per cent oxygen over a period of three hours, conspicuous emotional changes developed that were characterized at first by overconfidence, elation and ideational fixations and after an hour or so passed over into a feeling of dullness, lethargy, headache and lack of emotional restraint. Experienced pilots flying without oxygen at 12,000 feet have frequently given testimony of similar strain, irritability, discomfort and bad judgment.

If such grave changes are manifest at 12,000 feet, one naturally wishes to know what other functions may be impaired at the same time, and how soon in the course of ascent the changes can be recognized. Little by little, evidence is accumulating that vision, especially night vision,[14, 15] becomes conspicuously impaired. The gunner begins to lose accuracy of aim even at 8000 feet; the reaction time of the pilot in response to radio signals, or in facing danger in combat, is increased between 5000 and 10,000 feet, varying markedly with individual fliers.

In the cardiac sphere, White[16] reports on electrocardiograms taken on 45 supposedly normal persons exposed to altitudes as high as 20,000 feet with varying rates of ascent (see also Hoff[9]). The changes in the electrocardiogram, especially in the T wave, were noted in all subjects at about 5000 feet, the changes being conspicuous if the rates of ascent were rapid. U waves also became prominent with increasing anoxia, and since all changes disappeared on administration of oxygen, White concludes that myocardial anoxia is the cause of the alterations. As a result of his studies, White recommends that, to secure physiologic normality in a pilot, oxygen should be taken at altitudes above 5000 feet and that it should be mandatory in any flight exceeding 7500 feet. Similar changes in the electrocardiogram of animals have recently been observed by Wiggers[17] and in unpublished studies in this laboratory.

The use of oxygen in the lower altitude ranges has also become an established practice among high-altitude squadrons for the sake of washing out nitrogen, which in the higher altitude ranges is likely to give rise to "bends." Benson[18] finds that in normal subjects there is no change in the electrocardiogram if pure oxygen is breathed up to 30,000 feet.

Oxygen mask. Oxygen masks have been the subject of much discussion and study since the outbreak of the war. The B.L.B. mask was the only one commercially available in the United States at the beginning of the present war, and the old pipestem method of administering oxygen was still in use. The pipestem has proved dangerous and difficult for high-altitude operations, because it prevents the pilot from speaking and

is likely to drop from his mouth in the event of a serious emergency. All opinion, therefore, is in essential agreement that a face mask must be used for oxygen administration.

The original B.L.B. mask, both the nasal and the auronasal, had, for pilots, disadvantages that do not apply to patients lying prone in bed, for whom the masks were originally devised. One of the most serious difficulties was that at higher altitudes the valves of the mask tended to freeze, and the mask itself tended also to freeze to the face and thus irritate the skin.

Many special masks have lately been proposed with a view to obviating these difficulties. Thus, Barach and Eckman[19] describe an improved nasal mask. Other masks are now undergoing service tests in this country and abroad, and it is hoped that one will be found answering the ideal specifications recently set forth by Poppen,[20] namely, that the mask must be comfortable to wear, that it must have provision for microphone communication, that its valves must not freeze at -50°C., and that some provision must be made to vary the supply of oxygen in accordance with the respiratory needs of the pilot as these change. The actual details of how these requirements are being met cannot be discussed here.

Oxygen supply. Oxygen tanks remain the chief source of supply in all combat ships. In large bombers having a personnel of eight or ten during flight, in which missions are long and the altitude high, the oxygen tanks add very considerably to the weight load, and hence various proposals have been made to manufacture oxygen in flight. It is not a military secret that no one of the proposals for separating oxygen in the air has so far proved satisfactory. The new aluminum alloy tanks, however, have very considerably diminished the weight load, but since they are more vulnerable to machine-gun penetration they probably form a greater hazard to the pilot and the ship than the older, heavier oxygen tanks.

Pressure-cabin planes have so far not proved feasible for light combat craft.

Air Embolism and Its Prevention

During the course of the present war, certain pilots in high-altitude squadrons have experienced grave symptoms that could not be attributed to anoxia, acceleration or any form of metabolic disturbance. Susceptible persons, some at altitudes as low as 25,000 feet, developed paresthesias, with numbness and itching, sometimes restricted to the face, but often occurring sporadically all over the body. Later, aches and a sense of deep localized pressure often developed in one or more of the peripheral joints, especially the knuckles and wrists and occasionally also in other joints, particularly if there had been previous injury. These premonitory symptoms are followed sometimes quite quickly by the development of intolerable deep pain variously described as rheumatic or like that of a toothache, and often referred to a given joint or to a previously injured long bone — one of the best recent descriptions of this type of pain is that of Streltsov,[21] of the Pavlov Laboratories in Moscow. These intense deep pains are indistinguishable from those experienced by divers in the familiar syndrome of the "bends."[22] Recently, diving crews familiar with the bends have been subjected to decompression, and they have experienced at altitudes equivalent to 28,000 and 35,000 feet symptoms closely parallel with those which they had had when emerging too rapidly from an undersea dive.[23] Severe bends may be followed by collapse, pallor, sweating and respiratory difficulty, all developing with catastrophic rapidity. Such collapse has been thought to be due to sudden pulmonary air embolism.

Proof that high-altitude bends are caused by the liberation of nitrogen has been somewhat difficult to establish by direct observation, but the indirect evidence of similarity of symptoms is highly impressive (see Armstrong[24]). Quite recently, Walsh and Boothby[25] have found that bubbles begin to appear in the cerebrospinal fluid of human beings in a decompression chamber when subjected to a pressure equivalent to an altitude of 12,000 feet. The bubbles were fine, like those rising in good champagne. They became larger and more numerous at pressures approximating those of 28,000 feet, but after ten minutes' exposure to such altitudes the bubbles decreased in number and size, and later disappeared. The cerebrospinal-fluid pressure in these circumstances rose to a level 3 cm. higher than normal. This was believed to be due to the expansion of air bubbles.[26]

If bends occur as commonly as one might be led to suppose from the early reports, especially from observations made on men of older age groups, this condition is clearly a serious hazard for fliers. However, it has been found by Armstrong[24] and by many others who have lately investigated the problem that there is a large individual variation in susceptibility and that bends occur less commonly in the younger age group, for example, from eighteen to twenty-four, the average age of the combat flier, than in the older age group. Decompression-chamber tests suggest, moreover, that at least 50 per cent of a population of young male adults can withstand altitudes as high as 40,000 feet for periods of from one to six hours without developing bends. Hence it has been suggested that the high-altitude squad-

rons be selected on the basis of decompression-chamber tests.

Since bends are undoubtedly due to the formation of nitrogen bubbles in the tissues, an obvious suggestion would be to rid the tissues of their nitrogen by replacing the latter with helium[27] or by preoxygenation. This proposal has been studied by Behnke[28] and others. Armstrong and Heim,[29] for example, find that inhalation of pure oxygen from half to one hour before ascent prevents bends. But Whittingham,[30] in reviewing Armstrong and Heim's paper, mentions that observations in England have shown that inhalation of oxygen merely delays the onset of bends and does not actually prevent it. Furthermore, the recently published curves of Behnke and Willmon[23] indicate that at least five hours are required to eliminate nitrogen from the body by oxygen inhalation, and that owing to the fatty deposits in the marrow of the long bones, the bone marrow tends to hold its nitrogen more firmly than other tissues do. Since some at least of the deep pain associated with bends probably arises from bubble formation in the long bones, it would be essential to have at least five hours of preoxygenation to give a subject complete immunity from the distressing syndrome.

It is obvious, therefore, that from the point of view of combat flying it is not feasible to protect a flier by causing him to breathe oxygen for five hours before going up. But with careful selection of personnel for high-altitude operation, and with the delaying effects of pure oxygen from the ground up, bends cease to be a serious problem in military aviation.

Likewise for practical reasons, the use of helium to replace nitrogen has not been found feasible for combat pilots.

Anoxia of the Adrenal Glands[31]

In a little-quoted paper by Kellaway,[32] published in 1919, it was pointed out that a cat subjected for several hours to low-oxygen tension exhibits a rise of blood sugar and an increase of heart rate, both of which the author attributed to active secretion of the adrenal medulla: during decompression, paradoxical dilatation of the denervated pupil occurred, a phenomenon that had already been proved to be due to the liberation of adrenaline, since section of the splanchnic nerves completely abolished the dilatation. When the splanchnic nerves were sectioned, the blood-sugar elevation was abolished initially, but Kellaway encountered several animals, not fully accounted for in his protocols, in which after more prolonged exposure, the blood sugar actually rose.

Kellaway was not aware of the significance of the adrenal cortex, and it remained for Evans,[33] fifteen years later, to elucidate its action.* The latter observed a striking increase in the glycogen content of the liver and in the glucose level of the blood of rats exposed to half an atmosphere pressure in a decompression chamber, and he proved that the phenomenon failed to occur after adrenalectomy. He did not cut the splanchnic nerves, but he found that adrenocortical hormone restored the blood sugar and glycogen response in a specific manner. When exposed for twenty-four hours to the rarefied atmosphere, containing 10.5 per cent oxygen, the glycogen content of the liver of a normal animal increased ten to twenty times above its resting level, and the blood glucose increased markedly. Since the total carbohydrates in the animal had become augmented, it was evident that glucose must be formed from noncarbohydrate sources, probably entirely from proteins.

These observations have more recently been confirmed by a number of investigators, notably by Long et al.[36] and by Thorn and his collaborators.[37] Armstrong and Heim[38] found that hypertrophy of the adrenal glands occurs when animals are exposed repeatedly to low partial pressures of oxygen, suggesting that some substance is elaborated in the adrenal cortex when an animal is adjusting to anoxia. Strong confirmatory evidence pointing in this direction came in 1937 from Giragossintz and Sundstroem,[39] who pointed out that it took ten times as much adrenocortical extract to maintain adrenalectomized rats at half an atmosphere as it did at sea level. Very little clinical evidence is as yet available concerning the behavior of the adrenal cortex in man exposed to high altitudes, and it is not yet clear whether the administration of adrenocortical hormone in man facilitates his adjustment to high altitude or improves his performance. The results recently reported by Kendall[40] suggest that the corticosterone fraction rather than the desoxycorticosterone is responsible for the carbohydrate changes and possibly, therefore, for the adjustment to altitude. Armstrong[24] has suggested that the physical collapse which occurs in combat pilots after excessive exposure to diminished oxygen pressure may be due to atrophy of the overworked adrenal glands, the syndrome being essentially similar to that of Addison's disease. Huddleson and McFarland[41] have found that the administration of adrenocortical hormone is beneficial in cases of psychasthenia.

*Observations bearing on the reaction of the adrenal glands in anoxia, but in which no clear distinction is made between the adrenal cortex and the adrenal medulla, are found in the papers of Binet and Lanxade[34] and Houssay and Rietti.[35]

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 27331

PRESENTATION OF CASE

First Admission. A thirty-eight-year-old, Irish-born, single domestic was admitted to the hospital complaining of joint pains.

Two and a half years before admission, the patient began to suffer from attacks of arthritis characterized by redness, heat, swelling and limitation of motion in the various joints. Her feet, shoulders and sternoclavicular joints were first involved, and later her knees and the small joints of the fingers and hands. These attacks lasted for long periods, and to control the pain she took Oxyliodide, a proprietary drug containing cinchophen.

One year before admission, the patient became progressively more tired and noticed an increasing jaundice of the skin, without pruritus. Vomiting ensued, the stools became pale, and drowsiness was so pronounced that she slept most of the time. Abdominal distention developed, and a paracentesis was performed on three occasions. Severe nosebleeds occurred, and at the time of admission the patient had lost 20 pounds. She was in another hospital the best part of three months, and during this time the joint pains were in abeyance. Her strength and appetite gradually returned, and the jaundice faded; five months before entry, however, the joint pains returned.

The family history was irrelevant. Seven years before admission, the patient had had "jaundice," for which she was treated in another hospital. Nosebleeds had occurred for several months before the onset of her illness two and a half years previously.

Examination showed a poorly nourished, slightly jaundiced woman with minimal generalized adenopathy. There were signs of fluid at the right base. The heart was not enlarged, but a systolic murmur was audible; the blood pressure was 150 systolic, 80 diastolic. The abdomen was protuberant, with shifting dullness, and a small umbilical hernia was noted. The liver was palpable two fingerbreadths below the costal border, and the spleen was easily felt. The fingers were tapering and spindled; the ankles, elbows and knee joints were swollen, tender and limited in motion.

The temperature, pulse and respirations were normal.

The urine was normal. The stools were guaiac positive on one occasion but negative on six other examinations. Examination of the blood showed a red-cell count of 3,360,000 with 60 per cent hemoglobin, and a white-cell count ranging from 3400 to 6400, with a normal differential. The blood film showed moderate hypochromia, anisocytosis and poikilocytosis. The blood Hinton reaction was negative. The blood nonprotein nitrogen and uric acid were normal. The serum protein was 4.7 gm. per 100 cc. The serum van den Bergh was normal indirect, and the icteric index 4. The Takata–Ara test was strongly positive, and the bromsulfalein excretion showed 10 per cent retention of the dye. The blood sedimentation rate was 0.43 mm. per minute. Gastric analysis showed no free fasting acid but did show 10 units after histamine; a guaiac test was positive. A sugar-tolerance test was normal, the fasting value being 85 mg. per 100 cc. The basal metabolic rate was +19 per cent. An electrocardiogram showed slight left-axis deviation.

X-ray films of the involved joints showed narrowing of the joint space, irregularity of the articular surface, new-bone formation about the margins and marked decalcification. The Graham test showed the presence of a small stone within a poorly functioning gall bladder. Examination of the colon, sinuses and jaws was negative save for a single incisor-root abscess. A film of the chest showed a high right diaphragm, a slightly dull right cardiophrenic angle and a slight shift of the heart to the right.

During a three months' stay in the hospital, the patient's oral temperature frequently reached 100°F. On one occasion, she was given 3 gr. of Nembutal and ¼ gr. of morphine, with the result that she slept for three days and her temperature rose to 102°F. The patient was maintained on a regimen of high vitamins and physiotherapy, but responded so poorly that she was discharged to a hospital for chronic patients. On one occasion, an abdominal paracentesis yielded 6500 cc. of light-yellow fluid.

Final Admission (five years later). During the intervening five years, the joint symptoms and disability did not progress, but the patient had slight recurrent ascites and jaundice from time to time. She was readmitted complaining of nonradiating subxiphoid postprandial pain, usually followed by nonprojectile vomiting and unrelieved by food or milk. For two weeks, she had been unable to keep anything down, even water. The vomitus was never blood stained, and there was no change in bowel habits, or blood by rectum.

Examination was unchanged, except that the liver edge was no longer palpable (one observer excepted) and that there were no signs of ascites. The blood pressure was 130 systolic, 80 diastolic.

The temperature, pulse and respirations were normal. The urine was normal except for +++ test for bile on one occasion. The blood was normal. The stools were guaiac positive twice in five determinations. The blood chemical findings showed normal vitamin C, uric acid, protein and bilirubin levels; bromsulfalein excretion was normal. The prothrombin time was 33 seconds, the normal being 20 seconds. The corrected sedimentation rate was 0.3 mm. per minute. The Congo red test showed 50 per cent remaining in the blood at one hour. Gastric analysis showed no free acid. An electrocardiogram showed, as before, a moderate left-axis deviation.

X-ray films of the joints showed progression in the pathologic process and considerable bone atrophy, especially of the shoulder girdle. Examination of the gastrointestinal tract was suggestive of esophageal varices and demonstrated a 2.5-cm. by 1.0-cm. ulcer crater in the antrum, the center of the crater lying 3.5 cm. proximal to the pylorus. The appearance was that of a benign ulcer.

The patient was started on an ulcer regime and physiotherapy. The prothrombin time having risen to 55 seconds, she was given synthetic vitamin K, with a return to 31 seconds. The gastric symptoms disappeared, but a film taken two weeks after the first showed no diminution in the size of the ulcer.

One month after admission, the patient developed photophobia, with conjunctival injection, and corneal ulcers were noted, so that she was transferred to another hospital for further care. After a week's stay, during which the keratitis did not improve, being ascribed to riboflavin deficiency, her temperature rose suddenly to 101°F., and she became slightly disorientated and incoherent. The following day, the temperature was 102°F., and she was transferred back to the medical service of this hospital.

Examination showed an acutely ill woman lying flat in bed, with labored respirations, slight cyanosis of the lips and a dry tongue. The heart and lungs were unchanged. The blood pressure was 100 systolic, 60 diastolic. The abdomen was markedly swollen, and there was sagging in the flanks. Spasm was present throughout, and compression of the umbilical hernia produced a tinkling, gurgling sound. Peristalsis was absent. The patient expired a few minutes after examination.

Differential Diagnosis

Dr. Alfred O. Ludwig: In this case, we are dealing with a woman who obviously had chronic progressive rheumatoid arthritis and several complications that might go with that disease or with its treatment. The first important statement in this history is that she took a drug containing cinchophen. It is quite well known that cinchophen occasionally produces subacute or acute yellow atrophy of the liver. Although such sensitivity is infrequent, it is important to recall that of 190 cases reported up to 1933, 46 per cent were fatal. It is probable that many cases are unreported, so that the incidence of toxicity may be much more frequent. Because of the serious nature of such reactions, we no longer use the drug in this hospital. One should remember that, as in this case, toxicity may not develop until several months after the taking of the drug, and that it may also occur with small doses, one case being reported following a single dose of 7.5 gr. This woman developed the typical clinical picture of subacute yellow atrophy, with drowsiness, ascites, jaundice and an increased bleeding tendency. During the time she was jaundiced, she apparently had a remission of the joint symptoms. This phenomenon has been previously reported, particularly by Hench* of the Mayo Clinic, who points out that the quantity, not the quality, of the jaundice, is important. He states that a blood bilirubin level of 8 mg. or more per 100 cc. is necessary before much improvement in joint pain occurs. Such remissions may last anywhere from five weeks to one and a half years; then usually, as in this case, the joint pain returns. It is of further interest that this patient had jaundice seven years before admission and some four years before she began to take cinchophen. There is some evidence that cinchophen may be more toxic to patients who have had previous liver damage, regardless of cause.

Examination showed a slight generalized adenopathy that might very well have been a part of the rheumatoid arthritis. Adenopathy is not infrequent in rheumatoid arthritis and may also be associated with splenomegaly. I believe the fluid at the right base was secondary to the acute disturbance in the liver. It is quite common in such cases, and is probably due to irritation of the pleura over the diseased organ. The patient obviously had ascites, again secondary to the liver disease. The

*Hench, P. S., Bauer, W., Dawson, M. H., Hall, F., Holbrook, W. P., Key, J. A., and McEwen, C. The problem of "rheumatism" and arthritis: review of American and English literature for 1938. *Ann. Int. Med.* 13:1837-1990, 1940.

enlarged spleen was undoubtedly associated with the hepatitis rather than with the arthritis. The examination of the joints is characteristic of rheumatoid arthritis. The guaiac-positive stools are related to an increased bleeding tendency from liver disease. That is also true of her anemia and leukopenia. A lowered serum protein is quite frequently associated with liver disease, although it may also be found in long-standing cases of rheumatoid arthritis. At the time she arrived at the hospital, the acute process in the liver had subsided considerably, because the van den Bergh was normal, and the icteric index only 4. A positive Takata–Ara test confirmed the diagnosis of liver disease. This test depends on the presence of an increased amount of globulin in the blood. It is usually positive only with a severe degree of liver disease. The bromsulfalein test showed 10 per cent retention, which is also consistent with the diagnosis of liver disease. The sedimentation rate was just at the upper level of normal by our method. The patient had a somewhat low fasting sugar value, which again may be related to the liver disturbance.

I think it might be worth while to see the x-ray films.

DR. TRACY B. MALLORY: There is no x-ray man here, unfortunately.

DR. LUDWIG: In the absence of expert opinion, I shall have to limit myself to the findings in the joints. I should like to have some help in determining the character of the lesion in the stomach.

The joint findings are characteristic of chronic rheumatoid arthritis. The irregularity and narrowing of the joint spaces and the marked atrophy of the bones, with evidence of joint destruction and subchondral cysts are consistent with rheumatoid arthritis.

There were several examinations for esophageal varices, and I gather that they were thought to be present.

DR. MALLORY: Dr. Aubrey O. Hampton, on interpreting the films, thought they probably were present but could not be certain.

DR. LUDWIG: I am led to believe that they thought the ulcer was benign. Is that correct?

DR. MALLORY: It is, so far as the fluoroscopic findings and films are concerned.

DR. LUDWIG: Her marked overreaction to Nembutal and morphine is also of diagnostic value. We have observed such sensitivity to morphine in a number of patients with proved hepatic cirrhosis. In such cases, even small amounts of morphine may cause death.

The diagnostic problem here is to decide what caused death. From the history, I think we must conclude that this patient did have toxic cirrhosis of the liver, almost certainly secondary to the ingestion of cinchophen. However, the symptoms before her last admission seem to have been due to the presence of a gastric ulcer. The laboratory examination showed an increased prothrombin time, which is consistent with the liver damage. It is of some importance that the gastric analysis showed no free acid whatever at that time. This is unusual but not unheard of with benign gastric ulcers. It may be significant that the liver decreased in size over five years.

The Congo red test showed 50 per cent retention in the blood, and the test was probably carried out as part of an investigation of the incidence of amyloidosis in rheumatoid arthritis. This complication may occur as part of the disease, usually in chronic cases of long duration or in children with the severe Still's type. A loss of 20 to 30 per cent from the blood in one hour may be considered normal. Here, the loss of 50 per cent may be significant and may mean that this patient had a degree of amyloidosis. I do not believe that the eye lesions were secondary to the rheumatoid arthritis, although iritis occurs in 5 per cent of the cases in some series. Scleromalacia perforans is occasionally seen with rheumatoid arthritis and is thought to be due to the formation of rheumatic nodules on the sclera. It is possible that vitamin deficiency played a part in this case, owing to an inability to absorb these substances normally.

The final episode is poorly described. I cannot escape the conclusion that this patient died of general peritonitis. If that is so, perforation of the ulcer would be the most likely cause; however, one would expect some history of pain. At the time she re-entered this hospital, she obviously had fluid in the abdomen. The statement that there was a tinkling sound on compression of the hernia may mean that there was also free gas in the peritoneal cavity. This would be further evidence that a viscus had perforated, in this case the stomach. The ulcer may have been malignant. The absence of free acid is slight confirmatory evidence of malignancy, and the fact that the ulcer did not decrease in size over a period of two weeks is additional evidence. I do not know how we can decide that question other than by microscopic examination. Gastroscopy apparently was not done.

Are there any other conditions that should be considered? One might think of acute hepatic failure, but I do not believe the evidence given here allows such a diagnosis. The patient did not become jaundiced again, and there are none of the other characteristic findings of acute hepatic

failure. One wonders, with the previous history of stone in the gall bladder, whether she might have had an acute cholecystitis with perforation of the gall bladder; but again the evidence does not allow us to conclude this. If a gallstone played any part, she might also have had acute pancreatitis, but again the evidence is lacking. I shall make diagnoses of chronic rheumatoid arthritis, toxic hepatic cirrhosis and an acute perforation of a gastric ulcer, with general peritonitis. Microscopically, there may be some degree of amyloid disease as well.

DR. CHARLES L. SHORT: I saw this patient several times, and I think she was once a living example, and is now a dead example, of the dangers of cinchophen — the United States Food and Drug Administration has taken recognition of the dangers of cinchophen and aminophyllin and has placed certain restrictions on their use. She was disabled for the remainder of her life following cinchophen intoxication. The fact that she developed an ulcer is of some theoretical interest because acute gastric ulcers can be readily produced in dogs that have been given toxic doses of cinchophen. In fact, it is much easier to produce an ulcer in dogs after cinchophen than it is to produce liver damage. Whether her ulcer had any relation to cinchophen is quite doubtful. I do not know of any other cases in man that have been reported.

DR. WYMAN RICHARDSON: Is it true that in your material here the incidence of ulcer and liver disease is a commoner combination than that due to chance?

DR. MALLORY: I cannot say that it is. I thought for a time that it was, but as we see more cases, such a combination does not come up very frequently.

CLINICAL DIAGNOSES

Gastric ulcer.
Cirrhosis of the liver.
Rheumatoid arthritis.

DR. LUDWIG'S DIAGNOSES

Rheumatoid arthritis, chronic.
Toxic cirrhosis of the liver.
Gastric ulcer, with perforation.
General peritonitis.

ANATOMICAL DIAGNOSES

Chronic rheumatoid arthritis.
Toxic cirrhosis of the liver (probably due to cinchophen).
Benign peptic ulcer, with perforation.
General peritonitis.
Pneumoperitoneum.
Splenomegaly.
Esophageal varices.

PATHOLOGICAL DISCUSSION

DR. MALLORY: Dr. Ludwig has made this very complicated case seem almost absurdly easy. This patient showed an extremely small liver; it weighed only 650 gm., which was also the weight of the greatly hypertrophied spleen. Esophageal varices were present and very readily demonstrated at autopsy. There was no evidence, however, that they had bled at any recent period. There was a large ulcer in the stomach just at the edge of the prepyloric area. It measured 3 cm. in length, 1 cm. in width and 1 cm. in depth. We were very suspicious that it might prove to be malignant, and therefore cut a considerable number of microscopic sections through its margins, all of which showed a perfectly benign mucosa. We must accept it as a benign ulcer in spite of the fact that this patient showed so very little hydrochloric acid in the gastric secretions. The test was positive on only one occasion, and then weakly. The final episode was a perforation of the gastric ulcer, and there was generalized peritonitis, with considerable amounts of air beneath the diaphragm, which showed up clearly on post-mortem x-ray plates. There were, of course, all the usual changes of a severe chronic rheumatoid arthritis.

A PHYSICIAN: Was there any amyloid disease?

DR. MALLORY: No.

CASE 27332

PRESENTATION OF CASE

A thirty-four-year-old woman entered the hospital complaining of the sensation of a lump in her throat.

The patient felt well until two years before admission, when the sensation of a lump in her throat appeared just above the suprasternal notch. It was constant, felt like "a piece of apple" in her throat, and was aggravated by swallowing, although there was no difficulty in swallowing or in breathing. The symptom disappeared within three weeks but returned from time to time when the patient was overworked, tired and nervous.

Four months before admission, the sensation once again became constant and grew worse, producing slight difficulty in swallowing if food was taken too quickly. The patient stated, "The morsel seems to stick in my throat for a moment." Two months later, she saw her physician, and at

this time a constant substernal burning sensation had appeared; x-ray films of the chest were taken. Two weeks before admission, the patient stopped work, and immediately her symptoms subsided.

At no time had there been cough, hoarseness, dyspnea or weight loss. For the previous week, the patient had noticed a dull pain on movement of the left elbow, wrist and thumb. She had suffered from diphtheria and the usual childhood diseases. The family history was irrelevant.

On examination the patient was well developed and well nourished and in no apparent discomfort. The thyroid gland was palpable in the suprasternal notch during swallowing. The cardiac apex and left border of dullness were in the anterior axillary line. A blowing systolic murmur was heard at the apex and in the aortic area; the blood pressure was 120 systolic, 85 diastolic. Examination of the lungs, abdomen, pelvis and nervous system was negative.

The temperature, pulse and respirations were normal.

The urine was normal. The blood showed a red-cell count of 5,200,000 with a hemoglobin of 80 per cent, and a white-cell count of 10,600 with 67 per cent polymorphonuclears. A blood Hinton reaction was negative.

An x-ray film of the chest showed a round soft-tissue mass approximately 6 cm. in diameter behind the heart in the angle between the spine and diaphragm on the left side. The esophagus touched its anterior wall, but was probably not adherent to it. The mass did not pulsate and showed no evidence of calcification. The base of the right upper-lung field was unusually bright, the heart was normal in position, and the spine showed no abnormalities in the region of the mass.

Films taken in the upright and horizontal positions, with the patient lying on the right side, failed to show definite change in the size of the mass.

An electrocardiographic recording showed normal rhythm at 90, with a PR interval of 0.14 second. There was a tendency to right-axis deviation. T_2 was low, T_3 slightly inverted.

On the fourth hospital day, an operation was performed.

Differential Diagnosis

Dr. Jacob Lerman: The story is that of pressure symptoms in the neck of two years' duration, with a sensation of a lump in the throat, but no hoarseness and no difficulty in breathing. There is no mention of an objective pressure symptom, namely, clearing of the throat, which the patient usually disregards. The symptoms became worse four months before entry, and in addition, difficulty in swallowing became definite. There is no mention of cough, hoarseness, dyspnea, pain in the chest or cardiac embarrassment. When the patient stopped work, the pressure symptoms subsided. On the whole, pressure symptoms in the neck fluctuate considerably, depending on such factors as nervousness, fatigue and rest.

Physical examination revealed no discomfort, and the patient was able to breathe properly. The thyroid gland was palpable, but no mention is made of its size. The description is that of a substernal gland, because when she swallowed, only the top portion could be felt.

The x-ray report fails to mention the thyroid gland. It seems that if the gland were in the position described, the radiologist should have visualized it, particularly after a swallow of barium. There is no mention of this procedure. According to the x-ray examination, the heart is not displaced by the mass in the posterior mediastinum. We should expect some deformity of the trachea, not only from the mediastinal mass but also from the goiter that was felt in the suprasternal notch. However, there is no description of the trachea.

The electrocardiogram may be unimportant, or may indicate pressure on the heart by the tumor, producing slight right-axis deviation.

The differential diagnosis boils down to some form of mediastinal tumor. The first question I should like to settle, assuming it was neoplastic, is whether it was benign or malignant. It seems from the description that the tumor was not malignant. It is described as a discrete lump, which fits in with a diagnosis of benign tumor. The absence of tissue destruction, as indicated by freedom from pulmonary infection and toxemia, and the absence of venous obstruction, involvement of the recurrent laryngeal nerve or erosion of bone argue against malignancy. Of the benign tumors, one must consider neurofibroma, fibroma, chondroma, myoma, dermoid and simple cyst.

The evidence is against aneurysm. Presumably, the aorta was visualized and found to be distinct from the mass, which did not pulsate; the blood Hinton reaction was negative. One ought to consider a diverticulum of the esophagus, which could produce such a large mass. Certainly, in this event, x-ray study should have revealed a connection between the esophagus and the mass. Also, a large diverticulum would change in appearance with change in position. It occurred to me that a large diaphragmatic hernia could produce this picture. Here, also, the radiologist would have visualized the connection between the mass and

the stomach, and the mass would have changed in size and shape with change in position.

Because of the presence of a goiter, one must consider the possibility that an intrathoracic goiter had moved down from the usual position and had gradually wandered to the bottom of the thoracic cage. Any tumor of thyroid origin that lies below the superior strait has to descend as it grows. If it descends below the level of the arch of the aorta, it can grow laterally or posteriorly. The fact that the symptoms were intermittent suggests that the mass grew intermittently. This is consistent with the growth of a portion of a substernal goiter downward into the posterior mediastinum. I am assuming that the examiner found a substernal thyroid gland. Against this assumption is the lack of pressure symptoms. I should like to know more about the x-ray appearance of the esophagus and trachea.

Dr. Charles L. Short: I saw these x-ray films, and there was no sign of a substernal thyroid gland. I also examined the patient before x-ray examination was made and did not feel any goiter.

Dr. Lerman: The examiner was very explicit. Should I take his findings at their face value or be influenced by what Dr. Short has said? The position of the mass is consistent with intrathoracic goiter. I have seen similar tumors that have "plunged" into the thorax and assumed positions either lateral or posterior to the heart without producing much deformity of the adjacent organs. I shall have to consider, first, a large intrathoracic goiter and, secondly, and commoner, a benign tumor of the mediastinum, such as a neurofibroma. The x-ray report mentioned that the base of the right upper lung was unusually bright. This was probably due to compensatory emphysema of this lobe as a result of partial collapse of the right middle and lower lobes.

Dr. Short: I saw this patient before she had an x-ray examination, and she certainly gave the appearance and the story of a patient with functional symptoms. I was rather surprised when the x-ray report of mediastinal tumor came back.

There was one interesting thing in the physical examination — the apparent displacement of the heart to the left. I think that might have given us a clue to the presence of tumor in the chest. I should be interested to know whether the heart was actually displaced. The murmur was not enough to make a diagnosis of organic mitral regurgitation. The x-ray films that I saw were even more confusing, because the tumor mass was first interpreted as being cystic, the most probable diagnosis being a congenital cyst of the lung. On the basis of that and of the patient's story, it seemed unlikely that the mass was causing symptoms. I was rather hesitant about advising operation without having a period of observation, but she had the tumor removed.

Dr. Lerman: There is nothing here to indicate that the heart was displaced. It was normal on x-ray examination.

Dr. Short: Yes; but on physical examination, the apex was clearly palpable outside the mid-clavicular line.

Dr. Lerman: Did you mean displacement anteriorly?

Dr. Short: I just said displacement. I suppose it was anterior rather than to the left, but the apex was certainly felt outside the mid-clavicular line, and in the absence of intrinsic heart disease, I should assume that there was some mass in the mediastinum displacing the heart.

Dr. Edward D. Churchill: This case, like others we have recently discussed, shows the chaotic state of the interpretation of symptoms arising from mediastinal tumors, and is characteristic of those of a very large number of patients. The symptoms seem completely removed from anything that this tumor could possibly have done, and so remote, as Dr. Short has intimated, that one might be entirely convinced that they have nothing to do with a mediastinal tumor. I think that is often so. Frequently, as in this case, I have not been at all convinced that the mediastinal tumor has had anything to do with the symptomatology; yet the patients must be treated. There are sensations that do arise from tumor in the mediastinum, and patients not infrequently have great difficulty in finding words to express just what they are experiencing. It is not pain; it is not oppression; it is not covered by the usual words that doctors use to describe sensations. One asks them these questions, and they say, "No," but they eventually find a word for it. The result is that I have seen patients with malignant tumors of the mediastinum carried on for long periods as neurotics simply because they could not express what they were experiencing. I think the decision to operate must be based on the frank impossibility of knowing what type of tumor one is going to find and what that tumor is going to do for the next few years.

Clinical Diagnosis

Neurofibroma of mediastinum.

Dr. Lerman's Diagnoses

Intrathoracic goiter or mediastinal neurofibroma.
Partial collapse of right middle and lower lobes.

Anatomical Diagnosis

Neurofibroma of mediastinum.

Pathological Discussion

Dr. Tracy B. Mallory: This patient was explored, and a fibromatous tumor that apparently arose from the sympathetic chain was found. It lay just above the diaphragm and lateral to the aorta. It had pushed the lower lobe forward and had produced partial collapse. Histologically, it was a fibromatous tumor, with some areas of what we call "palisading," which suggest a neurogenous origin. It also showed extensive areas of xanthomatous degeneration, large parts of the tumor being bright yellow. On histologic examination, these yellow areas were composed largely of mononuclear phagocytes containing cholesterol, similar to those found in atheromatous plaques. The diagnosis of neurofibroma is not open to any question.

Dr. Ralph Adams: This patient was well from the time of operation, two years ago, until last fall. At that time, she was seen because again she had the sensation that a morsel was sticking in her throat, and she complained of nervousness, irritability and difficulty in swallowing. These symptoms had been precipitated by the persuasive arguments of a young man who wished to marry her. X-ray films were negative, and she was reassured about her general condition.

Dr. Lerman: Was there any comment at operation about where the thyroid gland was located?

Dr. Mallory: I do not think they got anywhere near it. The operative incision was low and posterior?

Dr. Churchill: Yes; it was well down.

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PREVENTIVE GERIATRICS

THE paper covering the potentialities of preventive geriatrics that appears in this issue of the *Journal* will have something of an emotional appeal to all but our youngest readers, for it embodies an outlook on advancing years that is generally cherished in middle age. That for which we all fondly hope as we reach the climacteric presents a pleasant prospect and makes good reading, even though we know it cannot be attained by all. That it may become more generally attainable, however, is not entirely a wistful hope but a reasonable, and perhaps even a scientific, goal for which to strive. Just as pediatrics has thrived on the irresistible urge of the baby to grow up, so may geriatrics promote itself by husbanding the natural involutional processes that tend to adjust and protect the earthly tabernacle against the ravages of time.

It is to be hoped that this promotion will not be forced, as it sometimes has been in political discussions that have moved along parallel lines. Much more knowledge of the physiology of age—here called gerontology—is needed before we can know how to improve our present habits. The conviction that they are not the best possible habits has long been entertained and expressed, as by Lord Rosebery:

> It is a black moment when the heralds proclaim the passing of the dead, and the great officers break their staves. But it is sadder still when it is the victim's own voice that announces his decadence, when it is the victim's own hand that breaks the staff in public. I wonder if generations to come will understand the pity of it!

It is to be hoped that statistical data will be carefully interpreted. With the rapidly increasing numbers of older people, as well as with our changing community and family habits, it is going to be easy to quote figures that, if they are not carefully interpreted, may become the basis of ill-advised public action. Decreases in death rates are bound to be balanced by subsequent increases in other categories of the *International List*. Part of these increases must therefore be counted as compensating phenomena, and reckoned among the current landmarks of the constantly changing scene. The years of our lives are still approximately three score and ten. We have not increased the natural span; we have merely prevented infectious and metabolic accidents from snuffing us out prematurely.

It is to be hoped that gerontology may set a stage upon which a rational concept of aging processes will develop. Too often, in the past, we have been tempted by such plausibles as medical glandular and surgical rejuvenation. What has become of the operations of Voronoff and Steinach? Such devices will fail for the same physiologic reason that Ponce de Leon failed in his quest for the Fountain of Youth. We badly need a basic science on which to build. For gerontology the field is open.

Lastly, it is to be hoped that practitioners of preventive geriatrics will not rush to establish for themselves examining and qualifying boards. None who is not a competent general practitioner can hope to become a competent geriatrician, and the time has come when a specialty for the general practitioner could have a salutary effect on medical values. Who better than the general practitioner could guide us across the falling ground and down into the valley? Those must be the years when the individual and family and community backgrounds will mean the most; those have been the years when the family doctor has served the best — let him now perfect himself. He is apter to have known us through all the seven ages described by the melancholy Jacques, and such knowledge is destined to be an important part of geriatrics.

> And so from hour to hour we ripe and ripe,
> And then from hour to hour we rot and rot,
> And thereby hangs a tale.

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UNITED CHINA RELIEF

THE Medical Division of United China Relief has announced the appointment of Dr. Co Tui as medical adviser to the China Defense Commission, which is in charge of all purchases for the Chinese government under the terms of the Lease-Lend Act. A total of $228,721 has been allocated for medical and relief supplies to free China; this sum was collected through the Bowl of Rice parties in California and through the tireless efforts of the national committee of United China Relief and the China Emergency Relief Committee. Dr. Co Tui, with the advice and help of leading American surgeons and physicians, will co-ordinate requests for medical provisions from medical experts in China and will help decide which supplies will most effectively meet Chinese needs.

The tremendous program of medical relief calls attention to facts that are frequently overlooked in the campaign of all-out aid to Britain: China, too, has for years been resisting the aggression of an Axis power, and the Chinese government and people deserve the fullest possible aid from the democracies. All-out aid to China is a contribution to the front line of American defense; medical aid to China will carry sorely needed relief not only to the inhabitants of Chungking, the most thoroughly bombed city in the world, but also to the millions who fight ceaselessly by day and night for the preservation of their country and the vindication of their national honor.

MEDICAL EPONYM

JARISCH–HERXHEIMER REACTION

Professor O. Jarisch (1850–1902), of Graz, discussing "Therapeutische Versuche bei Syphilis [Therapeutic Experiments in Syphilis]" in a series of articles, writes as follows in his first installment, in the *Wiener medizinische Wochenschrift* (45: 721–724, 1895):

> The following experiments were based on an observation that has certainly been made previously by many syphilologists, but has never to my knowledge been taken account of. I refer to the observation of a kind of reaction that shows itself in the first few days of a course of mercury inunctions in patients with syphilitic roseolae as an exacerbation of the topical phenomena of the disease. After from two to five inunctions or injections, the individual spots often become more distinct than before, and thus seem to be increased in number. This exacerbation of signs ends in an involution that, it seems to me, corresponds in rapidity to the intensity of the reaction.
>
> If, as indicated by numerous facts, the effect of mercury in syphilis is not due to a supposed *direct* antiparasitic effect, the preceding fact must suggest the conception that mercury has an (indirect ?) chemotactic, inflammatory effect analogous to tuberculin and the bacterial proteins.

Dr. Karl Herxheimer (b. 1861), chief physician of the Skin Department of the Municipal Hospital in Frankfurt am Main, and his assistant, Dr. Krause, contributed a paper, "Ueber eine bei Syphilitischen vorkommende Quecksilberreaktion [A Mercury Reaction Occurring in Syphilitic Patients]," in the *Deutsche medizinische Wochenschrift* (28²: 895–897, 1902). A portion of the translation follows:

> Some years ago, one of us observed striking changes in the exanthem of syphilis after the administration of mercury. More than a year ago, we made a similar observation of a patient suffering with a macular syphilide, which renewed our interest in the subject. The exanthem had changed to such a degree within about twenty-four hours after an initial inunction of 40 gm. of gray-mercury ointment that no characteristic of the original efflorescent, either of size, form or color, remained. The lesions were larger, had become elevated above the surface of the skin, had taken on

a bright-red color, and had rather the characteristics of an eruption of erythema exsudativum multiforme. After twenty-four hours, however, it had completely disappeared. . . . After we had observed the reaction in more than 60 cases and were in a position to study it, we believed that we could describe it as follows: It appears, as has been said, when a sufficient amount of mercury is completely absorbed for the first time. . . . If there is any single, distinctive feature of the phenomenon, it is the change in the syphilitic exanthem from a typical to an atypical form. . . . From what has been said, it seems justified to consider this a "reaction." This is developed by an oversensitiveness of the cell, which has been affected by syphilis toward mercury in such a way that mercury, by uniting with the cell, causes increased damage.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY*

Cesarean Section and Pneumonia Followed by Death

A thirty-two-year-old para II presented herself at a surgeon's office desiring a cesarean section. She stated that she was at term. Her desire for operation was based on unpleasant memories of the first labor and operative delivery, which the baby survived. Apparently no attempt was made to ascertain the history of the first pregnancy.

The past history was irrelevant, and the prenatal care during this pregnancy had been inadequate. Cesarean section, performed on a date set by the patient, was by the low-transverse cervical route. She was etherized by a nurse, and there is no indication in the history of how the anesthetic was taken. Death occurred nine days later, apparently because of a postoperative pneumonia. There was no autopsy.

Comment. This case is reported solely to emphasize the seriousness of needless cesarean section. Any laparotomy carries certain hazards, and even in the hands of trained anesthetists, pneumonia may cause death. A physician who does not appreciate this possibility is remiss in his obligations to his patients. In this case, there was no obstetric indication for cesarean section, and death should be attributed to lack of medical honesty.

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section. Letters should be addressed to Dr. Raymond S. Titus, *Secretary*, 330 Dartmouth Street, Boston.

CORRESPONDENCE

"ACCOUCHEMENTS FOR AMERICA"

To the Editor: We Americans are divided about many things, but interventionists and isolationists are agreed on one thing at least, and that is that America must prepare to defend itself. We hear cries for speeding up of manufacture of all sorts of vehicles and weapons of war. We are told that we need more ships, more tanks, more aeroplanes. I have as yet heard no one mention the fact that we shall need more young men to sail the ships, drive the tanks, fly the aeroplanes. In other words, America also needs more babies! Instead of other trite slogans, it might be well for American women in the position to do so to take "Accouchements for America" as their slogan. If Germany wins the war and proceeds to utilize the shipyards and other resources of Europe,—perhaps for an attack on this country,—it may mean that 1960 will be the year when our country's fate is at stake. In my opinion, we should do long-range thinking, and attempt to increase our potential army of that time right now. If our birth rate can be increased, we may be able to keep our place in a world that sees the dictators making frantic efforts to increase their populations.

We should not allow the so-called "newer races" to carry our burden in 1960. Most of these people have large families and are having their deliveries cared for by clinics. Apparently few of the wealthy, who can afford an unlimited number of children, are desirous of large families. I believe that physicians should co-operate to encourage large families among the other class—that is, the great independent, bill-paying middle class. In the last few years, hospitalization plans have helped.

We may soon have such things as a bachelor tax and subsidies given to young people to allow them to be married early. My reason for writing this letter is to suggest that we get the jump on the rest of the country and start the ball rolling by offering a definite plan to encourage new citizens.

The plan is as follows: each physician will announce to his patients that each couple's fourth delivery will cost only 75 per cent of the third; the fifth delivery 75 per cent of the fourth and so forth. For example:

Fee for third baby	$100
Fee for fourth baby	75
Fee for fifth baby	55
Fee for sixth baby	40
Fee for seventh baby	30

Those who object to this plan on the grounds that the fee is not commensurate with the time and trouble expended on the case should consider the service a patriotic duty, and reflect that, at the present time, innumerable French physicians would gladly accept even more burdensome conditions if France could be once again a strong, free, prosperous and happy nation. Incidentally, this movement will be an investment in good will, which will mean a great deal for our profession when the now apparently providentially postponed battle for socialized medicine is again commenced in the postwar period.

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